JOURNAL
of the
American Veterinary Medical Association

Formerly AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

OSCAR V. BRUMLEY, President
Columbus, Ohio

M. JACOB, Treasurer
Knoxville, Tenn.

EXECUTIVE BOARD

A. E. CAMERON, 1st District; E. P. ALTHOUSE, 2nd District
L. A. MERILLAT, 3rd District; WILLIAM MOORE, 4th District
H. C. H. KERNKAMP, 5th District; I. E. NEWSOM, 6th District
W. A. SULLIVAN, 7th District; L. J. ALLEN, 8th District
H. W. JAKEMAN, Chairman, 9th District; F. A. ZIMMER, 10th District
CHAS. W. BOWER, Member-at-Large
OSCAR V. BRUMLEY, ex officio; H. D. BERGMAN, ex officio

The American Veterinary Medical Association is not responsible for views or statements published in the JOURNAL, outside of its own authorized actions. Reprints should be ordered in advance. Prices will be sent upon application.

Vol. XCI, N. S. Vol. 44 November, 1937 No. 5

VETERINARY STUDENT ENROLLMENT FOR 1937-1938

Reports made by the deans of the twelve veterinary colleges in the United States and Canada show that the number of veterinary students enrolled this year is in excess of the number for last year by 271. This increase compares with 303 a year ago, 332 two years ago, and 316 three years ago. In other words, the number of veterinary students has been increasing at the average rate of more than 300 per year for the last four years.

It should be kept in mind that the figures representing veterinary student enrollment during recent years have included quite a number of pre-veterinary students. Eight of the twelve veterinary colleges report 549 students enrolled for pre-veterinary work this year. Four of our colleges do not recognize pre-veterinary students. Another fact to be kept in mind in this connection is that we have no way of determining how many pre-veterinary students are enrolled in educational institutions not included in the list of twelve veterinary colleges. It has been suggested that pre-veterinary students should not be enumerated in these annual reports on student enrollment. Having started to do so, we have continued to include them. However, the

figures are segregated in such a way that anyone can compute the number of regular students in any college or in all of them. Dean Dykstra, of Kansas State College, reports that the pre-veterinary students at Manhattan have been transferred from the Division of Veterinary Medicine to the administrative control of the Division of General Science. The six-year courses in Animal Husbandry and Veterinary Medicine and General Science and General Medicine have been discontinued.

If we look at the figures representing the students enrolled in the regular four-year veterinary course, we find that there are 2,170 such students. These are fairly well distributed throughout the four classes, there being 657 freshmen, 528 sophomores, 542 juniors and 443 seniors. Incidentally, it may be pointed out that the number of seniors enrolled this year is the largest for many years, and indicates that the number of 1938 graduates will be the largest for quite a while.

Another fact that becomes apparent from an examination of the accompanying table showing the distribution of students in the twelve veterinary colleges is that there has been a very noticeable tendency for the enrollment in eleven of the colleges to even up. This is a direct result of some of the colleges being compelled to place limitations on the number of veterinary students enrolled. This has resulted in a large number of prospective veterinary students being declined admission by these institutions. Many of these students have sought and obtained admission elsewhere. Dean Hagan, of Cornell, reports that the total number of applications considered by the Admissions Committee this year was 276. His report shows 40 freshmen enrolled. It would be interesting to know what became of the 236 students who were declined.

Last year, we reported that six colleges had found it necessary to restrict the enrollment of veterinary students. This year Dean Giltner, of Michigan, reports that the Governing Board of Michigan State College has limited the enrollment of veterinary students in a single class to 64. The figures for the East Lansing institution indicate that both the freshman and sophomore classes contain the maximum number. Dr. C. D. McGilvray, Principal of the Ontario Veterinary College, writes that it was necessary to restrict the enrollment in the freshman class this year to residents of Canada.

For the year 1926-27, Texas had two freshmen and nine other students in the three classes. This year, there are just 100 first-year students at College Station. For the year 1926-27, Alabama had 17 veterinary students in all four classes. This

year, there are 157 regular veterinary students at Auburn, with 53 pre-veterinary students reported. For the year 1926-27, Washington had 42 veterinary students enrolled. This year, there are 239 at Pullman, including 92 pre-veterinary students. For the year 1926-27, Michigan had a total of 28 students in the four classes. This year, there are 203 students in these classes, besides 44 pre-veterinary and seven graduate students. For the year 1926-27, four of the larger colleges had about 52 per cent of the regularly enrolled students. This year, these four colleges have only about 35 per cent of the total enrollment.

It is encouraging to note an increase in the number of veterinarians taking graduate work this year. The number is the largest in five years but should be larger. In all probability the 38 graduate students reported by six colleges do not represent the entire number of veterinarians taking postgraduate work.

Veterinary student enrollment for the college year 1937-1938

College	Fr.	Soph.	Jun.	Sen.	Spec.	Grd.	Tot.	1936–37	Chg.
Alabama Poly. Inst.	53	8	62	34	53*	4	214	191	+ 23
Colorado State Coll.	41	40	33	16	65*	0	195	161	+ 34
Cornell University..	40	42	40	31	0	12	165	157	+ 8
Iowa State College..	60	56	48	48	81†	6	299	253	+ 46
Kansas State College	58	57	64	41	61*	1	282	279	+ 3
Michigan State Coll.	64	64	33	42	44*	7	254	228	+ 26
Montreal, Univ. of..	16	14	13	10	0	0	53	49	+ 4
Ohio State Univ....	69	52	45	55	74*	6	301	310	— 9
Ontario Vet. College	60	66	60	55	0	0	241	219	+ 22
Penna., Univ. of....	55	38	53	30	13	0	189	194	— 5
Texas A. & M. Coll.	100	52	66	39	81*	2	340	237	+103
Washington, S. C. of	41	39	25	42	92*	0	239	223	+ 16
Totals (1937–38)...	657	528	542	443	564	38	2772	2501	+271
Last year (1936–37)	629	506	470	322	553	21	2501	2198‡	+303

*Pre-veterinary students.
†Includes 79 pre-veterinary students.
‡Total number of students for 1935-36.

TOO FEW OR TOO MANY?

Now that figures are available for study, let us look at the situation confronting the veterinary profession in general and the veterinary colleges in particular, as far as the problem of veterinary education is concerned. Have we enough veterinarians available to do the work expected of them? Are there more than are needed? Or is there a shortage of veterinarians?

No better proof of the importance of this question is to be found than the fact that at least four A. V. M. A. committees made some reference to this general subject in their reports made at the Omaha meeting.

Those who hold to the opinion that there are too many veterinarians seem to be in the minority. There are quite a few who seem to think that the situation is fairly satisfactory just as it is. On the other hand, the weight of opinion seems to lead to the conclusion that more veterinarians could be used if they were available.

A few years ago, the A. V. M. A. Committee on Education made an exhaustive study of the requirements for veterinarians and came to the conclusion that we would need about 300 new veterinarians each year for the next 50 years merely to maintain the numerical strength of the profession at the 1930 level. It was stated that there should be a total of 1,400 students in the colleges, divided as follows: 400 freshmen, 350 sophomores, 350 juniors and 300 seniors. These figures were approximated for the year 1933-1934, when the colleges (including the two Canadian institutions) reported 331 freshmen, 338 sophomores, 387 juniors and 314 seniors.

According to the 1937 report of the Committee on History, our colleges have graduated about 2,450 veterinarians during the past decade. This is approximately 500 less (average of 50 per year) than the estimated requirements for replacements, to say nothing of the additional veterinarians needed for the expanding activities of the profession.

The following paragraphs are taken from the 1937 report of the Committee on Policy:

> Speaking before a state association the other day, a state veterinarian remarked that outstanding projects had to be abandoned from lack of man power. Qualified applicants for admission to veterinary schools are being turned away by the hundreds. This Committee could read into this record letter after letter showing the futility of young men applying for admission. This appears to indicate that the colleges have taken over a responsibility they are unable to fulfill for the people of this country.
>
> We speak loosely about expanding milk and meat inspection to every community; of supplying medical service to every flock of poultry; of restricting the use of biological products to graduates of the veterinary schools, when, as a matter of fact, there is not personnel enough to justify any expansion whatsoever. So, the expansion of the veterinary service must begin in the colleges before this Association can rationally recommend the development of new fields. Otherwise, the handling of animal diseases will pass into new hands.

To get the full import of these paragraphs the entire report should be read.

Much has been said about veterinarians participating in public-health programs. The present situation is summed up in one paragraph taken from the 1937 report of the A. V. M. A. Special Committee in Food Hygiene:

> Any analysis of veterinary participation in existing or contemplated milk inspection activities inevitably leads to the problem of trained personnel. In view of our overtures for appropriate recognition of veterinarians in the various fields of public health, we must face the fact that "there is not a sufficient professionally trained veterinary personnel in this country to supply the veterinary service needed." This statement, from the 1936 report of the A. V. M. A. Committee on Public Relations, has implications far beyond the limited scope of your Special Committee on Food Hygiene. It is substantiated by the experience of members of this Committee who have had opportunities during the past year to place veterinary graduates in milk control work but were unable to find veterinarians to accept.

We must not get out of step. Our efforts would be largely wasted if we were to get, let us say, a meat inspection law in every state not now having one and then have no veterinarians available for the positions created by these laws. Last month the JOURNAL carried an announcement of an examination for veterinary meat inspectors in California. This stated that the usual residential requirements would be waived, *because of the lack of veterinarians in California qualified for this type of work.*

DEATHS FROM SULFANILAMIDE

Much space in the press recently has directed attention to one of the weak spots in the federal Food and Drug Law. In an effort to capitalize on the publicity given sufanilamide during the past year, a large manufacturing pharmaceutical house placed on the market a so-called elixir in which sufanilamide was dissolved in another compound known as diethylene glycol. This "elixir of sulfanilamide" was given wide distribution through the drug trade. In Tulsa, Oklahoma, nine deaths followed its use. Then five persons in East Saint Louis, Illinois, died after taking the "elixir." Up to November 1, over 50 fatalities were reported to the health authorities throughout the country.

The latest information would indicate that the solvent, diethylene glycol, used in preparing the "elixir," has been largely responsible for the trouble. It is quite likely that some of the deaths attributed to the product will, upon proper investigation, be found to have been caused by something else. However, a bad situation has been created, and we are reminded of similar occurrences in the past, when dangerous drugs were allowed to get into the hands of those who knew little or nothing about them.

In commenting on the situation, W. G. Campbell, Chief of the Food and Drug Administration, gave it as his opinion that "these unfortunate occurrences may be expected to continue because new and relatively untried drug preparations are being manufactured almost daily at the whim of the individual manufacturer, and the damage to the public health cannot be accurately estimated." Mr. Campbell further stated that "the only remedy for such a situation is the enactment by Congress of an adequate and comprehensive national Food and Drugs Act, which will require that all medicines placed upon the market shall be safe to use under the directions for use, and for patent drugs and new drugs a federal licensing system is probably the answer."

EXECUTIVE BOARD ELECTION

A special election is now being held in A. V. M. A. Executive Board District 7 (Washington, Oregon, Idaho, Montana, Wyoming, North Dakota, South Dakota, Nebraska, Alaska, Hawaii and the Philippine Islands) for the purpose of selecting a member of the Board to fill the vacancy caused by the removal of Dr. C. H. Hays from Pierre, S. Dak., to Lansing, Mich.

Ballots asking for nominations have been sent to all members in District 7 who are in good standing. These ballots were dated September 15, 1937, and, in conformity to the provisions of the By-laws, the polls will remain open for 60 days. In other words, the polls will not be closed until November 15. As soon as possible after that date, ballots for the election proper will be mailed to all A. V. M. A. members in District 7 who are in good standing. The ballots will contain the names of the five members receiving the first, second, third, fourth and fifth highest numbers of nominating votes.

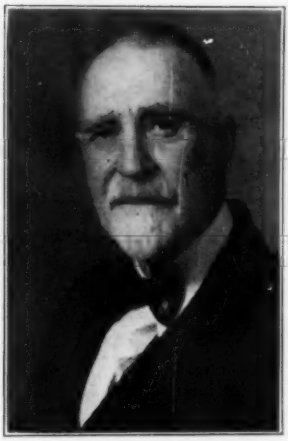
THE 1939 CONVENTION

Attention is directed again to the fact that the Executive Board of the American Veterinary Medical Association will consider invitations for the 1939 convention at the winter meeting of the Board in Chicago. The dates for the meeting have been fixed for November 28 and 29. The meeting this year is being held a few days earlier than usual, in order to avoid conflicts with the meetings of the Official Research Workers in Animal Diseases of North America (November 30) and the United States Live Stock Sanitary Association (December 1-2-3). Any members who desire to extend invitations on behalf of their cities can secure further information by communicating with the Secretary.

Meet—

DR. H. W. JAKEMAN

Of Boston, Mass. Chairman of the Executive Board of the A. V. M. A. Represents District 9 (New York and New England States).

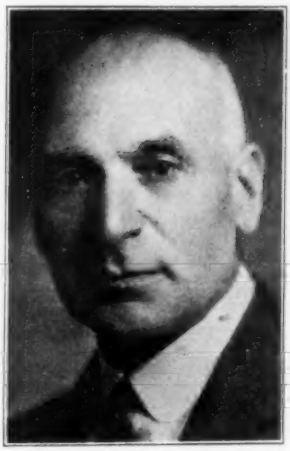


DR. JOHN L. TYLER

Of Whittier, Calif. Elected First Vice-President of the A. V. M. A. at the convention recently held in Omaha.

DR. H. D. BERGMAN

Of Ames, Iowa. President-elect of the A. V. M. A. Professor and Head of Veterinary Physiology and Pharmacology, Iowa State College.



DR. A. E. CAMERON

Of Ottawa, Canada, now serving his second term on the Executive Board of the A. V. M. A., representing Canada.

APPLICATIONS FOR MEMBERSHIP

The months immediately following each annual convention are usually rather lean months as far as applications for membership go. It is not difficult to find an explanation for this. As a matter of fact, there are several factors that operate to produce this condition. The "urge" of the convention has spent its force for the time being, and the next meeting is a long way off. This year, however, one phase of the situation has been changed. With the new custom of electing our presidents one year in advance of taking office, it has been possible to step up the work of making appointments and notifying appointees. During the past month all resident secretaries were notified of their appointment. This gives the resident secretaries a chance to get an earlier start on their membership campaigns and the additional time so afforded should be productive of even better results than in the past.

(See July, 1937, JOURNAL)

FIRST LISTING

ANDERSON, MARVIN E. Route 1, Burlington, Wash.
B. S., D. V. M., State College of Washington, 1934
Vouchers: R. R. Weller and C. E. Sawyer.

CASWELL, DON L. State Game Farm, Mason, Mich.
D. V. M., Iowa State College, 1937
Vouchers: B. V. Alfredson and C. F. Cairy

CORDY, DONALD R. New York State Veterinary College, Ithaca, N. Y.
D. V. M., Iowa State College, 1937
Vouchers: Herbert L. Gilman and R. R. Birch.

DEHNER, LORIS A. Concordia, Kan.
D. V. M., Kansas State College, 1936
Vouchers: E. J. Frick and E. R. Frank

FRY, LT. LLOYD V. Veterinary Hospital, Fort Snelling, Minn.
D. V. M., Iowa State College, 1937
Vouchers: Maj. J. G. Fuller and Lt. L. W. Hibbs

JENKINS, LESTER E. 3014 44th Ave. S., Minneapolis, Minn.
D. V. M., Chicago Veterinary College, 1917
Vouchers: Joab P. Foster and John S. Dick, Jr.

KNORTH, CHARLES A. Red Wing, Minn.
D. V. M., Chicago Veterinary College, 1913
Vouchers: Joab P. Foster and John S. Dick, Jr.

LEE, THORVALD M. Watertown, Minn.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: Joab P. Foster and Donald B. Palmer

MOYS, MURREL D. 204 State Bldg., Los Angeles, Calif.
B. S., D. V. M., State College of Washington, 1929
Vouchers: W. L. Curtis and A. G. Feers.

NIEMEYER, WILLIAM E. 1533 Crenshaw Blvd., Los Angeles, Calif.
B. S., D. V. M., State College of Washington, 1935
Vouchers: W. L. Curtis and A. G. Feers.

ROBERTSON, LT. COL. DANIEL S. Fort William McKinley, Rizal, P. I.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: Lt. Col. Clell B. Perkins and Maj. Seth C. Dildine.

WARREN, DAYTON M. 232 S. 6th E., Missoula, Mont.
D. V. M., Ohio State University, 1924
Vouchers: W. J. Butler and H. F. Wilkins.

Applications Pending

SECOND LISTING

(See October, 1937, JOURNAL)

Anderson, Lt. Everett T., Box 776, Tuscaloosa, Ala.
Anderson, Robert J., Jr., 503 U. S. Court House, Fort Worth, Texas.
Crawford, Lt. Edwin M., Fort Snelling, Minn.
Cyrog, Robert J., Ohio State University, Columbus, Ohio.
Fisher, Lt. Le Roy T., 63 Colonial Ave., Trenton, N. J.
Folsom, Richard H. Plum City, Wis.
Manogue, Lawrence J., Seaford, Del.
Omohundro, Lt. Richard E., 2308 Bryan Park Ave., Richmond, Va.
Pang, Wilson M., 2200 Star Rd., Honolulu, T. H.
Rosenfe'd, Benjamin, 828 Mechanic Rd., Osage, Iowa.
Tax, Herman, State Institute of Applied Agriculture, Farmingdale, L. I., N. Y.
Winchester, Lt. Ben N., 1 E. University Pkwy., Baltimore, Md.

The amount which should accompany an application filed this month is $5.83, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.83, the additional $5.00 being for the 1938 dues.

Civil Service Examination

The U. S. Civil Service Commission will announce early in November a non-assembled examination for the position of Senior Veterinarian (Director of the Regional Laboratory for the Study of Animal Diseases, at Auburn, Alabama). Those interested in this examination should immediately communicate with the U. S. Civil Service Commission, Washington, D. C., and request an announcement of the examination which will give the educational requirements and necessary experience requisite to qualifying for the position.

State Board Examination

Connecticut State Board of Veterinary Registration and Examination. State Office Building, Hartford, Conn. January 4, 1938. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

The Seventh World's Poultry Congress will be held at Cleveland, Ohio, July 26 to August 3, 1939.

COMING VETERINARY MEETINGS

San Diego County Veterinary Medical Association. San Diego, Calif. November 9, 1937. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

Hudson Valley Veterinary Medical Society. Albany, N. Y. November 10, 1937. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Willamette Valley Veterinary Medical Association. Chamber of Commerce Rooms, Forest Grove, Ore. November 10, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. November 17, 1937. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Mississippi Valley Veterinary Medical Association. Galesburg Club, Galesburg, Ill. November 17, 1937. Dr. Lyle A. Gray, Secretary, Bushnell, Ill.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. November 17, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Kansas City Veterinary Association. Auditorium, General Hospital No. 1 Annex, Kansas City, Mo. November 15, 1937. Dr. C. W. Schulz, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. November 24, 1937. Dr. M. W. Allam, Secretary, Media, Pa.

Horse and Mule Association of America. Palmer House, Chicago, Ill. December 1, 1937. Mr. Wayne Dinsmore, Secretary, 407 S. Dearborn St., Chicago, Ill.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. December 1, 1937. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. December 1, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

National Association of B. A. I. Veterinarians. Hotel La Salle, Chicago, Ill. December 1-3, 1937. Dr. F. A. Imler, Secretary, Box 187, Kansas City, Kan.

United States Live Stock Sanitary Association. Hotel La Salle, Chicago, Ill. December 1-3, 1937. Dr. L. Enos Day, Secretary, 3933 Drexel Blvd., Chicago, Ill.

Houston Veterinary Association. Houston, Texas. December 2, 1937. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, 515 W. Church Ave., Knoxville, Tenn. December 4, 1937. Dr. R. E. Bauch, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. December 7, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. December 8, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Western Michigan Veterinary Medical Association. Greenville, Mich. December 9, 1937. Dr. Chas. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. December 14, 1937. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Nebraska State Veterinary Medical Association. Grand Island, Neb. December 14-15, 1937. Dr. J. D. Sprague, Secretary, David City, Neb.

Western New York Veterinary Medical Association. Buffalo, N. Y. December 16, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

South Dakota Veterinary Medical Association. Cataract Hotel, Sioux Falls, S. Dak. December 16-17, 1937. Dr. G. E. Melody, Secretary, Gettysburg, S. Dak.

American Association for the Advancement of Science. Indianapolis, Ind. December 27, 1937-January 1, 1938. Dr. Henry B. Ward, Secretary, Smithsonian Institution Bldg., Washington, D. C.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 3-7, 1938. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.

Pennsylvania, Conference of Veterinarians at University of. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. January 4-5, 1938. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

Maine Veterinary Medical Association. Wooster House, Hallowell, Me. January 5, 1938. Dr. S. W. Stiles, Secretary, Falmouth Foreside, Me.

Cornell University, Annual Conference for Veterinarians at New York State Veterinary College, Ithaca, N. Y. January 6-7, 1938. Dr. W. A. Hagan, Dean, New York State Veterinary College, Cornell University, Ithaca, N. Y.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 6-8, 1938. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Minnesota State Veterinary Medical Society. Nicollet Hotel, Minneapolis, Minn. January 10-11, 1938. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 10-11, 1938. Dr. F. Y. S. Moore, Secretary, McAlester, Okla.

Texas, State Veterinary Medical Association of. Houston, Texas. January 13-14, 1938. Dr. M. B. Starnes, Corresponding Secretary, Public Health Dept., Dallas, Texas.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 18, 1938. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 18-20, 1938. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Kansas Veterinary Medical Association. Jayhawk Hotel, Topeka, Kan. January 19-20, 1938. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Iowa Veterinary Medical Association. Des Moines, Iowa. January 18-20, 1938. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Ohio State Veterinary Medical Association. Neil House, Columbus, Ohio. January 20-21, 1938. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Mississippi State Veterinary Medical Association. Jackson, Miss. January 24-25, 1938. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.

Michigan State College Short Course for Veterinarians. Michi- State College, East Lansing, Mich. January 24-28, 1938. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

STUDIES ON NUTRITIVE EFFICIENCY OF COMMERCIAL DOG FOODS*

By AARON ARNOLD† *and* C. A. ELVEHJEM
Department of Agricultural Chemistry, University of Wisconsin, Madison, Wis.

Comparatively few studies on the nutritional requirements of dogs are reported in the literature. This is mainly due to the fact that their requirements follow the general rules of nutrition suggested for other mammals. In addition, dogs are not used extensively as laboratory animals in nutrition studies because of the time and expense involved in preparing sufficient amounts of purified rations for such large animals.

During the past few years, there has been a tremendous increase in the production and use of commercially canned dog foods. It has been assumed that all the nutritional requirements of dogs can be met safely and adequately by a product prepared through routine canning of animal and plant materials. The extent to which the products on the market meet these requirements is a matter of considerable practical as well as scientific interest. A study of products of this nature should also give some indication of the extent to which an industry manufacturing such products has benefited or can benefit from extensive nutritional studies. It is only in recent years that the importance of the minerals and vitamins, in addition to the recognized factors, the proteins, fats and carbohydrates, in nutrition has been disclosed and appreciated. A knowledge of the existence of these factors and methods for their determination have given us more definite information concerning the building of nutritionally complete diets.

Although the final evaluation of any supposedly complete diet depends upon the demonstration that the diet will produce good growth, normal development, and reproduction in the species for which the food was intended, at least preliminary information can be gained through the use of small laboratory animals. Certain conclusions are reached in a much shorter period of time by the use of the albino rat, which has proved to be of such tremendous importance in animal nutrition studies. More recently, the chick also has been shown to be a very valuable laboratory

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937. Published with the permission of the Director of the Wisconsin Agricultural Experiment Station.

†Wilson and Company Fellow.

animal. Since many of the results with both rats and chicks have been shown to apply directly to human nutrition, there is no reason to believe that results with these animals will not also give valid information in the case of dogs.

EXPERIMENTAL

A number of canned dog foods were obtained in case lots on the open market. Although they were picked at random, many of them were the more expensive brands and representd a large proportion of the total volume sold.

Method of feeding: The dog foods were removed from the cans and dried overnight at 50° C, in front of the fan. This precaution was taken to reduce the destruction of the labile factors to a minimum. The material dried at any one time was always fed within a week or less after drying. The products were fed in dry form in order that food consumption records could be kept.

Care of rats: The rats were kept in cages supplied with half-inch mesh, wire-screen bottoms. The food was kept before the rats at all times and placed in cups, which prevented scattering of the feed. Water also was available at all times.

The animals were obtained from our stock colony and placed on experiment when about 25 days of age, at which time they weighed between 40 and 50 grams. The animals were weighed weekly.

Care of chicks: Day-old White Leghorn chicks, with an initial weight of about 35 grams, were divided into groups of four and placed in cages equipped with wire-screen bottoms and suitable warmers. The dried dog foods and water were kept before the chicks to permit *ad libitum* feeding. Weights were recorded weekly.

Results of feeding dried dog food as sole article of diet: The data recorded here have been in process of collection over a period of three years. A number of the products were assayed at various times within this period without appreciable differences beyond the minor ones expected in the variation between lots prepared at different times.

The results of feeding 17 brands of dog food, each representing a separate concern, to rats are shown graphically in figure 1. Since the rates of growth of males and females are significantly different, the growth curves of each are plotted. Samples A586, A580, A507, A551, A573, A554 and A508 may be considered first-grade foods. The appearance of the animals fed these brands was excellent. The remaining eleven samples ranged from borderline to poor in quality. Samples A572, A557, A563 and A559 were not

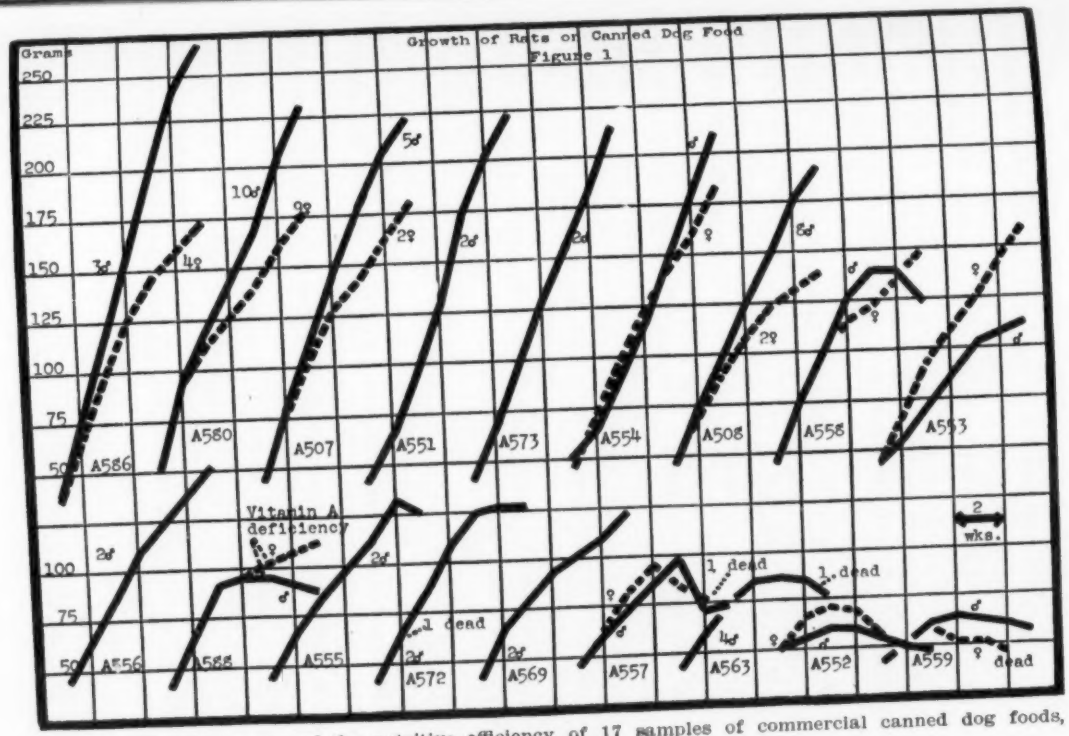


FIG. 1. Biological assay of the nutritive efficiency of 17 samples of commercial canned dog foods, with rats.

able to sustain the life of the rats during the six weeks of the experiment. These might more accurately be described as deficiency foods. The rats on sample A588 developed symptoms characteristic of vitamin-A deficiency during the fourth week. Since the animals that died on samples A572, A557, A563 and A559 did not show specific deficiencies, the only conclusion that can be drawn is that these were deficient in more than one nutritional essential.

When the above dried foods were fed to chicks, results of the same general order were obtained. Some of the typical results are given in figure 2.

The chicks on some of the samples showed characteristic symptoms of polyneuritis. This demonstrates that the main deficiency in some of the products at least is a lack of vitamin B_1. This deficiency was not so evident in rats because it is more difficult to produce this type of a deficiency in rats.

The comparative efficiency of the different samples is further emphasized in table I, in which is tabulated the average daily gain for the male and female rats and the grams of dried food required to produce 1 gram gain in weight.

The daily gain of the male rats fed samples A586, A580, A507, A551, A573, A554 and A508 for the six-week period is 3.5 grams or better per day, with an outstanding value of 5.5 grams per day for sample A586. The females show correspondingly good growth rate in the neighborhood of 3.0 gm per day.

The animals fed the first seven samples needed 4.0 gm or less to make a gram gain during the last five weeks on experiment. The first week is not used in these calculations in order not to penalize any foods that were not consumed in sufficient amounts the first day or two. The efficiency of the remaining eleven samples varied widely and these were significantly lower in value.

To gain a clearer view of the problems involved in the preparation of a nutritionally complete dog food, it is necessary to dispel some of the illusions that have been presented in the literature and consider the requirements individually.

PROTEINS

The nutritive value of any protein is directly dependent upon the amino-acids which this protein can supply. Whether the number of essential amino-acids is ten, due to the additional amino-acids recently disclosed by Rose, or more, due to requirements not disclosed by short-time growth experiments, is immaterial. Two conditions must be met: first, that the proteins must be hydrolyzable by body enzymes and, second, that the essential

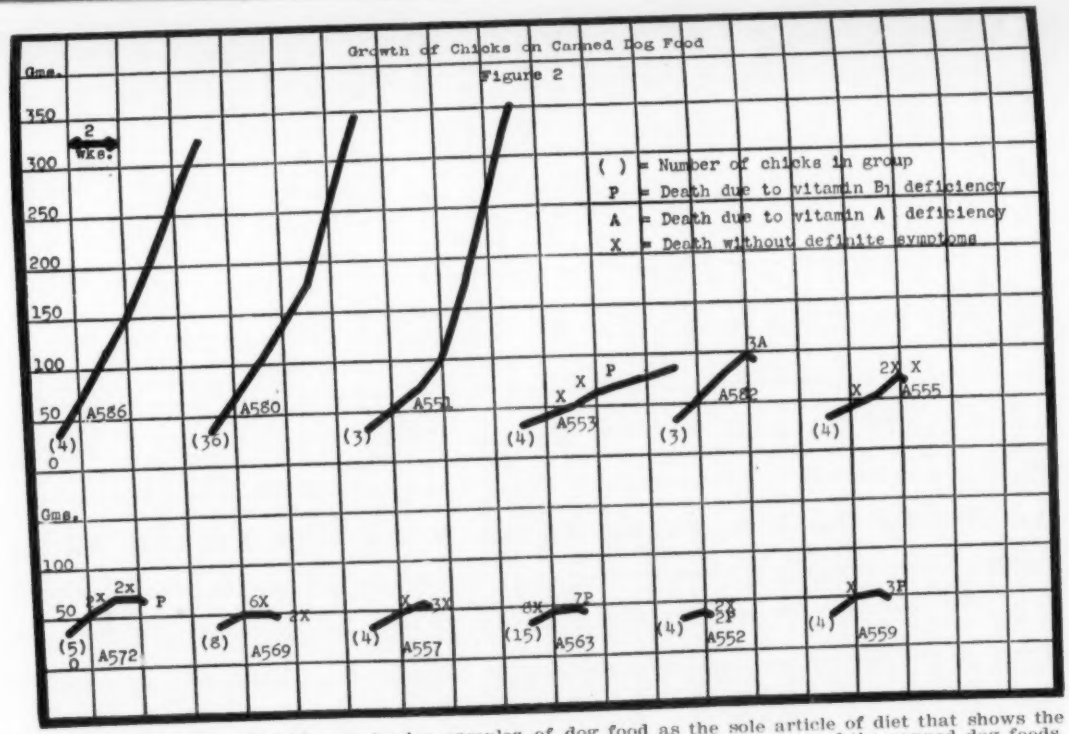


FIG. 2. Growth of chicks on twelve samples of dog food as the sole article of diet that shows the high incidence of polyneuritis caused by a low Vitamin B_1 content in certain of the canned dog foods.

TABLE I—*Comparative efficiency of different samples.*

SAMPLE	RATS	SEX	WEIGHTS		GAIN PER DAY (GM)	GM FOOD PER GM GAIN 2-6 WKS. INC.
			INITIAL	6 WKS.		
A586	3	M	40	269	5.5	
	4	F	36	173	3.3	3.3
A580	10	M	51	232	4.3	
	9	F	52	177	3.0	3.3
A507	5	M	44	223	4.3	
	2	F	43	181	3.3	3.5
A551	2	M	42	223	4.3	3.9
A573	2	M	41	215	4.1	4.0
A554	1	M	52	210	3.8	
	1	F	46	184	3.3	3.6
A508	8	M	46	192	3.5	
	2	F	46	139	2.2	3.4
A558	1	M	45	124	1.9	
	1	F	46	149	2.5	5.8
A553	1	M	43	113	1.7	
	1	F	45	160	2.7	4.6
A556	2	M	45	153	2.6	5.9
A588	1	M	40	88	1.1	
	1	F	43	114	1.7	6.6
A555	2	M	45	125	1.9	5.9
A572	2	M	40	128 (1 d.)	2.1	4.6
A569	2	M	41	123	2.0	4.8
A557	1	M	45	73	0.7	7.2
	1	F	47	(1 d.)		
A563	4	M	42	77 (1 d.)	0.8	10.0+
A552	1	M	51	48	.....	.....
	1	F	51	49	.....	.....
A559	1	M	42	57		10.0+
	1	F	43	(1 d.)	0.4	

d. = dead.

amino-acids must be present in adequate quantity. Since most dog food manufacturers take the precaution of including a number of protein sources, both of these conditions probably are fulfilled. Certain protein sources considered alone, such an animal organs, casein, and soy bean proteins have been shown to be very good. However, when two or more protein sources are used, there is no method of predicting the value of the mixture aside from feeding trials or analyses of the proteins for the amino-acids, methods too laborious for routine work. Mixtures of vegetable sources are appreciably improved by the addition of meat proteins.

Since the amino-acids are generally considered stable to processing, products made from an original mixture containing a variety of materials should show no protein deficiency. The important question, therefore, is the total amount of protein which the food should contain.

It is interesting to note that of the six samples showing excellent growth, all but one contained approximately 10 per cent protein (canned product). Two or three of the samples showing very poor growth contained only 5 to 6 per cent. Of course, it is impossible to conclude that the poor growth was related to poor protein. However, we can say that canned dog foods containing 10 per cent protein will give excellent growth providing the other essential nutrients are present. Whether the protein can be safely reduced much below 10 per cent remains to be determined.

Fat and Carbohydrate

The practice of manufacturers is generally not to pay particular attention to the fat and carbohydrate content of the dog food. There is no scientific objection to this practice, since the ratio of fat to carbohydrate in the diet may be varied within wide limits.

Ivy[1] has reported that dogs are able to digest starch. The chief objection to rations high in carbohydrate is that carbohydrates displace minerals and vitamins, and thus act to decrease the percentage of these factors in the diet. Ivy has also noted that dogs are unable to tolerate diets containing much over 40 per cent fat. The nutritive value of the carbohydrates and fats is generally considered to be unimpaired by processing.

Energy

For the purpose of correlating the total food intake with the vitamin requirements, it is necessary to discuss briefly the energy requirements of dogs.

Dechambre (quoted by Linton)[2] has compiled a table that lists the energy requirements of dogs. Brody, Procter and Ashworth[3] have published food maintenance prediction tables based on body weight which were calculated from studies on basal metabolism, endogenous urinary nitrogen, and neutral sulfur excretion.

Karr[4] and Cowgill[5] found that 70 to 80 calories per kilogram body weight per day maintained dogs that weighed in the neighborhood of 15 pounds for long periods of time. The simplified diet low in the factors of the vitamin-B complex (diet III) included casein 41 parts and fat 25 parts. Fifteen and three-tenths gm of this diet supplied 72.8 calories (1 kilo unit).

Salmon[6] has recently stated that one pound of dry ration (Alabama) was sufficient for a 40- to 50-pound dog.

The curves of Dechambre and of Brody, Procter and Ashworth have been charted in figure 3 for comparison. The values of Karr, Cowgill and Salmon cannot justifiably be extended to include the entire weight range of dogs, although the value submitted by Salmon has been charted in the figure. At the higher weights the amount of food necessary is less since the energy requirements do not increase in proportion to body weight but in proportion to surface area. It is seen that the maintenance prediction curves of Brody, Procter and Ashworth (figure 3), derived by directly converting Total Digestible Nutrients into terms of dog food, agree well with the results of the other investigators. A 15.4-pound dog (7 kilograms) would require 146 gm of dog food or about 584 calories, which is slightly higher than the 490 to 560 calories recommended by Cowgill for dogs of this weight. Table II also predicts 2.5 pounds of dog food for a 50-pound dog, or ¾ pound of dog food on the dry basis, which is in good agreement with veterinary practice.

During the periods of rapid growth and during pregnancy, dogs require more than maintenance allotments of food. Experiments in progress at the present time at this laboratory indicate that the energy needs of puppies are adequately met by doubling the predicted maintenance values. The increased demands of pregnancy and lactation must also be satisfied with increased allotments.

VITAMINS

The recent literature[2,7] has not attempted to state the vitamin requirements of dogs in terms of daily requirement. It is necessary that these levels be definitely established in order that dog foods may be made adequate in this regard. Furthermore, certain individual vitamins have been overestimated and the require-

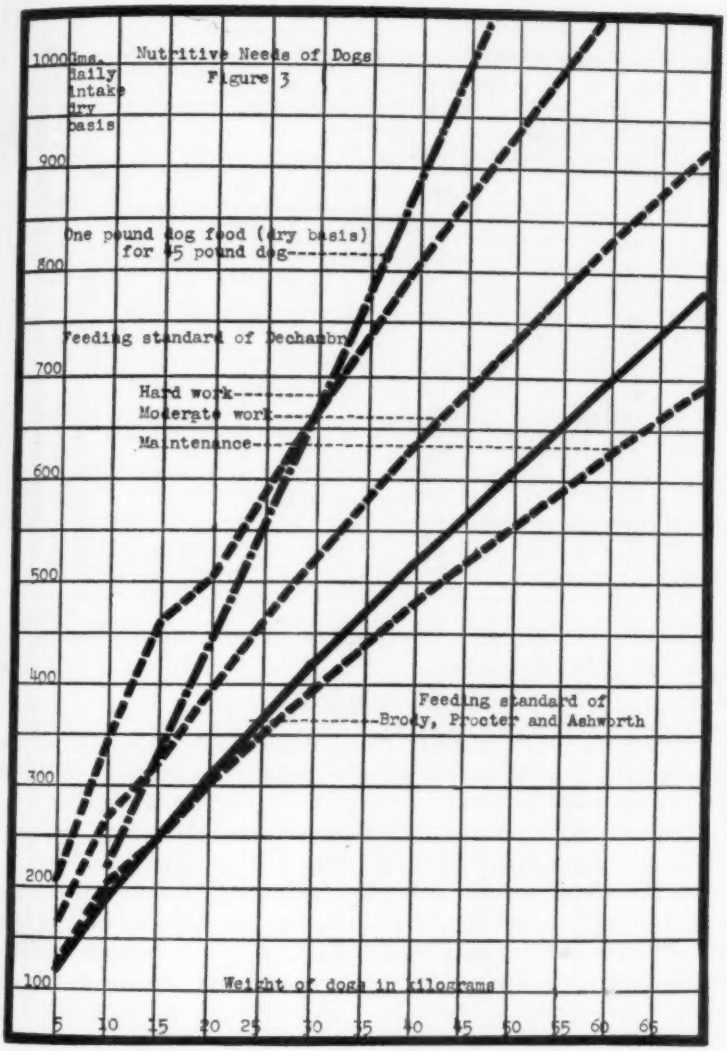


FIG. 3. A comparison of the nutritive needs of dogs according to Brody, Procter and Ashworth; Dechambre, and Salmon.

TABLE II—*Amounts of dog food (dry and fresh basis) for dogs of different weights.*

Body Weight (Lbs.)	Dog Food		
	Dry Basis (Lbs.)	Fresh Basis	
		Lbs.	Oz.
2	0.0723		3.85
3	0.0932		5.2
4	0.120		6.4
5	0.141		7.5
6	0.161		8.6
7	0.181		9.6
8	0.199		10.6
9	0.217		11.6
10	0.234		12.5
13.2	0.286		15.2
15.4	0.321	1	1.0
17.6	0.354	1	3.0
19.8	0.385	1	4.5
22	0.416	1	6.0
25	0.457	1	8.3
44	0.690	2	5.0
50	0.758	2	8.0
66.1	0.932	3	2.0
88.2	1.152	3	14.0
100	1.26	4	3.0

ments set at such high levels that the manufacturers tend to discount vitamins altogether.

VITAMIN A

Steenbock, Nelson and Hart[8] in 1921, reported that young dogs placed on a vitamin-A-free diet developed ophthalmia. These results have been repeated and extended by other workers.

That ample vitamin A may be preserved in canned dog foods is shown in figure 4. The procedure of Elvehjem and Neu[9] was used, since it affords a rapid and accurate method for the biological assay of vitamin A. Day-old chicks were placed on the basal ration with supplements of the dog food to be tested added at different levels. The chicks on the basal ration without any supplement died during the fourth and fifth weeks, exhibiting the typical symptoms of a vitamin-A deficiency. The incorporation of 5 per cent of sample A586 into the ration was sufficient to protect all of the chicks from a vitamin-A deficiency for six weeks. When 10 per cent of the same product was added to the basal ration, improved growth resulted. Sample A588, which failed to protect rats from a vitamin-A deficiency, also allowed a vitamin-A deficiency to develop in chicks. Day-old chicks, placed on sample A588 as the sole article of diet, died of a vitamin-A deficiency

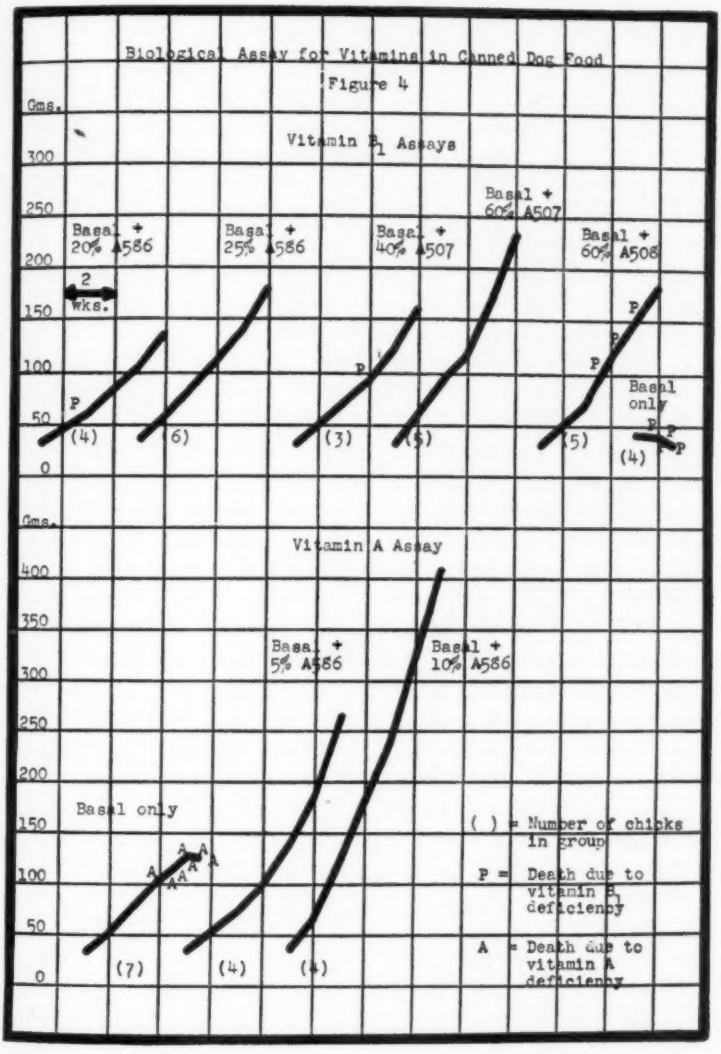

Biological Assay for Vitamins in Canned Dog Food
Figure 4
Gms.
Vitamin B1 Assays
Basal + 20% A586
2 wks.
Basal + 25% A586
Basal + 40% A507
Basal + 60% A507
Basal + 60% A508
Basal only
Gms.
Vitamin A Assay
Basal + 5% A586
Basal + 10% A586
Basal only
( ) = Number of chicks in group
P = Death due to vitamin B1 deficiency
A = Death due to vitamin A deficiency

FIG. 4. Biological assay for vitamins A and B_1 in commercial canned dog foods.

after three weeks, as shown in figure 2. It is thus seen that this factor is definitely of significance in canned dog food. Since rancid fats have been shown to inactivate vitamin A,[10, 11] as well as vitamin E, this may have been the reason for the lack of vitamin A in sample A588, a product high in fat.

The amount of vitamin A necessary for dogs has not been fully investigated. Frohring[12] has reported that puppies placed on a synthetic, vitamin-A-free milk lost 300 to 400 USP units of vitamin A per day, as judged by the Blue Units disappearing in the livers. On the basis of curative studies, Frohring reported that the minimum curative dose for puppies was 200 USP units of vitamin A per kilogram of body weight.

Crimm and Short[13] found that the vitamin-A requirement of the dog is between 22 and 47 I.U. per kilogram body weight per day. Guilbert, Miller and Hughes[14] correlated the data for rats, swine and cattle and have concluded that 6 to 8 micrograms of vitamin A or 25 to 30 micrograms of B carotene (pro-vitamin A) per kilogram body weight per day are sufficient to protect against hemeralopia (night-blindness), one of the earliest symptoms of vitamin-A deficiency. These workers also conclude that from the studies of storage at different levels of intake in animals it appears that five to ten times the amount necessary to satisfy the minimal intake is a desirable minimum to set for practical purposes.

Since vitamin A is commonly added as cod-liver oil, the requirements suggested by the California workers (70 micrograms vitamin A per kilogram body weight) is probably a good figure to use for dogs. A dog of 50 pounds (22.68 kg) would therefore require 1,588 units of vitamin A per day. If a dog of this weight requires about 2.5 pounds of dog food, then each pound of dog food should contain 635 units of vitamin A. Since sample A586 supplies 2,160 USP units of vitamin A per can, the vitamin A requirement can be readily met by a good grade dog food.

Vitamin B Complex

Vitamin B_1: The antiberiberi factor, vitamin B_1, first disclosed by Eijkman in 1897, is the most labile to processing. Elvehjem, Sherman and Arnold[15] have reported that 80 per cent of the vitamin B_1 activity may be lost by processing meat products at 250° F. for one hour and 50 minutes. This fact is, therefore, of prime importance in the manufacture of dog food.

Additional evidence to show that vitamin B_1 is the limiting factor in many dog foods is presented in figure 2. Samples A553, A563, A552 and A559 were fed as the sole source of diet to day-

old chicks. Of the 15 chicks on sample A563, six exhibited head retraction, characteristic of vitamin B_1 deficiency, before death. Two of the four chicks on sample A552 and three of the four chicks on sample A559 also showed evidence of a vitamin-B_1 deficiency before death. Of the four chicks on sample A553, one showed the syndrome of a vitamin-B_1 deficiency, two died without showing definite symptoms, and one survived the six-week experiment. A number of chicks fed the other samples likewise died due to a deficiency of vitamin B_1 in the dog food. The conclusion to be reached is that this heat-labile vitamin has not been given sufficient consideration by the manufacturers.

Since vitamin B_1 has sometimes been named the "appetite factor," it is small wonder that dogs have been noted to lose their taste for some of the products offered them.

The studies of Cowgill[16] have shown that dogs require about 2 International Units of vitamin B_1 per kilogram body weight per day. As calculated above, if a 50-pound dog (22.68 kg) requires 2.5 pounds dog food per day, then the minimum daily intake should be 46 International Units. Each pound should contain a minimum of 19 to 20 International Units. Calculations from the assays shown in figure 4 show that sample A508 contained slightly less than 45 International Units vitamin B_1 per can, and sample A507 contained between 45 and 68 I. U. vitamin B_1 per can, and sample A586 contained between 108 and 135 I. U. vitamin B_1 per can. The chick assay method outlined by Elvehjem, Kline, Keenan and Hart[17] was used for the determination of the antineuritic vitamin.

P-P factor: The anti-pellagra factor of humans, first discovered by the work of Goldberger, in 1926, was designated as the P-P factor. Goldberger also presented evidence to show that absence of the same factor in the diet caused black tongue in dogs. These facts have been verified by Koehn and Elvehjem[18] and Fouts, Lepkovsky, Helmer and Jukes.[19]

The wide distribution of the factor in animal tissues insures an adequate supply of this dietary essential in those dog foods that contain an ample amount of meat products. The P-P factor is much lower in plant sources. As Elvehjem, Kline, Keenan and Hart[17] have shown, heat treatment in the presence of moisture does not appreciably affect the P-P factor. The work on this factor has not progressed to the point where the daily requirements can be stated.

Riboflavin: Sebrell, Onstott and Hunt[20] have submitted evidence to show that dogs restricted to diets deficient in the heat-

stable, water-soluble factor, riboflavin, lose weight and eventually die. At autopsy, the animals are seen to have the pathological syndrome of yellow livers.

The wide distribution of this pigment in plant and animal products makes it likely that it will be present in adequate amounts in dog foods.

Vitamin B_6: The anti-dermatitis factor of rats, vitamin B_6, is more abundant in plant than in animal products. The recent findings of Fouts, Lepkovsky, Helmer and Jukes[21] indicate that this factor is also essential for dogs. In the absence of this factor, dogs become severely constipated and exhibit various neurological symptoms including incoördination, involuntary muscle spasms and generalized convulsions. They die from the effects of severe microcytic hypochromic anemia unless treated with a preparation containing vitamin B_6.

The evidence with respect to the heat stability of this factor is conflicting. It is generally regarded as more stable to processing than vitamin B_1.

Factor W: The fifth factor of the vitamin-B complex, recently disclosed by the work of Elvehjem, Koehn and Oleson,[22] also is destroyed to some extent by processing. Its function has not been characterized aside from its growth-promoting properties and, therefore, its relation to processed dog food must await further work. Factor W is abundant in liver and other animal products. Good growth of rats fed canned dog food shows that it can be conserved.

VITAMIN D

Mellanby[23] clearly demonstrated that rickets may be brought about by a deficiency of fat-soluble vitamin in the diet. McCollum and his co-workers[24] demonstrated that the antirachitic factor was distinct from vitamin A.

It cannot be too strongly emphasized, however, that rickets is not dependent entirely upon a lack of vitamin D. The relative proportions of calcium and phosphorus in the diet are also important factors in the etiology of rickets. It must further be emphasized that the total phosphorus in the diet, as indicated by chemical analysis, does not yield a complete picture. The "anti-calcifying" factor of cereals, first recognized by Mellanby, has recently been correlated with high phytin in cereals.

Phosphorus in this compound is linked as phosphoric acid with inositol. This phosphorus would be included in a chemical analysis, but in the absence of the enzyme phytase, it is not available to the animal organism. Thus, available phosphorus must be distinguished from total phosphorus.

These facts may aid in explaining some of the discrepancies which are found in published work on the vitamin-D requirements of dogs. In a recent bulletin by the Fleischmann Laboratories, it is concluded that dogs may need over 270 USP units of vitamin D per kilogram body weight per day. Morgan[25] has stated that 1 gm of cod-liver oil per kilogram body weight per day did not furnish sufficient vitamin D to protect dogs completely from rickets. The cod-liver oil was 2 per cent of the diet used. Chicks receiving optimum amounts of calcium and phosphorus supplied as 1 per cent $CaCO_3$ and 1 per cent $Ca_3(PO_4)_2$, in addition to the phosphorus in the unheated grains, are protected from rickets when the ration contains 17.5 to 22.5 USP units of vitamin D per 100 gm of ration. This amount of the antirachitic vitamin would be furnished by 0.27 per cent cod-liver oil. Since chicks have a higher body temperature than dogs and grow at a more rapid rate than the latter, the requirements of chicks for vitamin D should be at least as great as are the requirement of dogs.

The latter results agree excellently with the findings of Kozelka, Hart and Bohstedt,[26] who stated that collies, fed a ration containing 1 per cent $CaCO_3$, 1 per cent $Ca_3(PO_4)_2$, and 5 per cent dried skim milk, grew from 4.5 to 15 kilograms in 19 weeks with complete protection from rickets when 15 to 20 USP units (6 Steenbock units) of vitamin D were administered daily. This figure, therefore, shows that in the presence of optimum amounts of calcium and phosphorus, 1 to 1.3 units of vitamin D per kilogram body weight per day is sufficient to protect a dog from rickets. Therefore, as stated above, if a 50-pound dog (22.68 kg) requires 2.5 pounds of dog food per day, this amount of dog food should contain 23 to 31 units of vitamin D. Each pound should contain a minimum of 10 to 13 USP units of vitamin D. Although vitamin D is stable to processing, this amount cannot be obtained in the food product unless some extra source of vitamin D, such as cod-liver oil or irradiated yeast, is added.

Vitamin E

The reproductive factor in nutrition is widely distributed in nature. Its stability to processing insures its presence in dog foods. Rancid fats, however, inactivate the vitamin and this fact should be kept in mind.

Minerals

The requirements of dogs for the mineral elements have not been sufficiently studied to permit accurate estimations (Palmer[27]). Since mineral requirements have been established

TABLE III—*Suggested mineral element requirements for dogs.*

ELEMENT	MINIMUM REQUIREMENT PER KILOGRAM BODY WEIGHT PER DAY	MINIMUM REQUIREMENT PER POUND DOG FOOD
Calcium	0.02 gram	0.2 gram
Sodium and chlorine	0.3 gram sodium chloride	3 grams sodium chloride
Copper	0.00006 gram	0.0006 gram
Iodine	1 microgram	10 micrograms
Iron	0.0004 gram	0.0004 gram
Magnesium	0.01 gram	0.1 gram
Manganese	0.00004 gram	0.0004 gram
Phosphorus	0.04 gram	0.4 gram
Potassium	0.08 gram	0.8 gram
Zinc	0.00006 gram	0.0006 gram

for human nutrition, some idea of the requirements for dogs may be gained from these figures (table III).

Eleven elements are considered essential at the present time. Of these, precautions must be observed with respect to only five.

Calcium and phosphorus are completely available to the animal when supplied as bone ash, or ground bone, a practice that is to be recommended. When other lime salts are added to supply the calcium, provision should be made to insure sufficient phosphorus, since, as Morgan[28] has pointed out, most rickets in dogs is of the low-phosphorus type.

Dog foods containing appreciable quantities of plant products must contain added salt, since plant sources are high in potassium and low in sodium, an effect which must be counterbalanced.

In most regions, the iodine content of the plant and animal products used for the manufacture of dog food is undoubtedly high enough to supply the need. In areas where the natural products are known to be low in iodine, special precautions should be taken to insure an adequate supply of this element.

The other elements are ordinarily considered to be furnished in adequate amounts by the natural plant and animal products. We have never observed rickets, nutritional anemia, or thyroid disturbances in the animals fed any of the dog foods which we used in our experiments.

SUMMARY

1. Eighteen samples of commercial canned dog food were tested on rats and chicks. Seven samples were found to be excellent in quality. The remaining eleven samples ranged from borderline to poor in quality.

2. The energy, protein, carbohydrate and fat requirements of dogs are discussed on the basis of these and other studies.

3. The minimum vitamin requirements in terms of one pound of canned dog food, fresh basis, have been calculated. These levels are 635 U. S. P. units of vitamin A, 20 I. U. of vitamin B_1, and 10 to 13 U. S. P. units of vitamin D per pound can. The remaining vitamins are discussed with respect to their distribution in plant and animal products and stability to processing conditions.

4. The minimum mineral requirements have been calculated. Precaution must be taken to insure an adequate supply of five: calcium, phosphorus, iodine, and sodium chloride.

REFERENCES

[1]Ivy, A. C.: Vet. Med., xxxi (1936), p. 145.
[2]Linton, R. G.: Vet. Med., xxxii (1937), pp. 86-95.
[3]Brody, S., Proctor, R. C., and Ashworth, U. S.: Univ. Mo. Res. Bul. 220 (1934).
[4]Karr, W. G.: Jour. Biol. Chem., xliv (1920), pp. 255-276.
[5]Cowgill, G. R.: Amer. Jour. Physiol., lxvi (1923), pp. 164-175.
[6]Salmon, W. D.: Ala. Poly. Inst. Leaflet 13 (1935).
[7]Emerson, G. A.: No. Amer. Vet., xvii (1936), 9, pp. 24-40.
[8]Steenbock, H., Nelson, E. M., and Hart, E. B.: Amer Jour. Physiol., lviii (1921), pp. 14-19.
[9]Elvehjem, C. A., and Neu, V.: Jour. Biol. Chem., xcvii (1932), pp. 71-82.
[10]Mattill, H. A.: Jour. Amer. Med. Asso., lxxxix (1927), pp. 1505-1508.
[11]Powick, W. C.: Jour. Agr. Res., xxxi (1925), pp. 1017-1026.
[12]Frohring, W. O.: Vet. Med., xxxii (1937), pp. 76-82.
[13]Crimm, P. D., and Short, D. M.: Amer. Jour. Physiol., cxviii (1937), pp. 477-482.
[14]Guilbert, H. R., Miller, R. F., and Hughes, E. H.: Jour. Nutri., xiii (1937), pp. 543-564.
[15]Elvehjem, C. A., Sherman, C. A., and Arnold, A.: Jour. Biol. Chem., cix (1935), pp. 29-30.
[16]Cowgill, G. R.: The Vitamin B Requirement of Man. (Yale University Press, New Haven, 1934), p. 47.
[17]Elvehjem, C. A., Kline, O. L., Keenan, J. A., and Hart, E. B.: Jour. Biol. Chem., xcix (1932), pp. 309-319.
[18]Koehn, C. J., Jr., and Elvehjem, C. A.: Jour. Nutri., xi (1936), pp. 67-76.
[19]Fouts, P. S., Lepkovsky, S., Helmer, O. H., and Jukes, T. H.: Proc. Soc. Exp. Biol. & Med., xxxv (1936), pp. 245-247.
[20]Sebrell, W. H., Onstott, R. H., and Hunt, D. S.: U. S. Pub. Health Rpts., lii (1937), pp. 427-432.
[21]Fouts, P. J., Lepkovsky, S., Helmer, O. M., and Jukes, T. H.: Jour. Biol. Chem., cxix (1937), p. 34.
[22]Elvehjem, C. A., Koehn, C. J., Jr., and Oleson, J. J.: Jour. Biol. Chem., cxv (1936), pp. 707-719
[23]Mellanby, E: Lancet, cxcvi (1919), pp. 407-412.
[24]McCollum, E. V., Simmonds, N., Becker, J. E., and Shipley, P. G.: Jour. Biol. Chem., xlv (1922), pp. 293-312.
[25]Morgan, A. F.: Univ. Calif. Pub. in Physiol., viii (1934), 7, pp. 61-106.
[26]Kozelka, F. L., Hart, E. B., and Bohstedt, G.: Jour. Biol. Chem., c (1933), pp. 715-729.
[27]Palmer, R.: Vet. Med., xxix (1934), p. 171.
[28]Morgan, A. F.: No. Amer. Vet., xv (1934), 8, pp. 24-31.

Old Mules

According to a report in a recent number of *Horse and Horseman*, J. C. Hoover, of San Angelo, Texas, has a working span of mules which are 43 years and 42 years old, respectively. Mr. Hoover raised them on his farm and has worked them together since they were colts.

DIAGNOSIS OF POULTRY DISEASES*

By S. H. McNUTT, *Ames, Iowa*
Iowa State College

This paper is divided into a first part, having to do with diagnosis in general, and a second part, in which some certain specific diseases are considered. Fowl leukosis and the respiratory diseases are not included because these are discussed elsewhere. Except for coccidiosis, the animal parasites and the ailments they cause are also excluded. The discussion applies particularly to conditions in the Midwest.

General Considerations

There are a number of factors that have an influence on poultry diseases and a knowledge of these factors often aids materially in a diagnosis and suggests methods of prevention. A knowledge of them is particularly helpful in diagnosis where laboratory facilities are not available. These conditions that influence disease also emphasize the importance of proper poultry husbandry.

A general discussion of the diseases of any particular type of animal is often begun by a statement that a knowledge of correct husbandry is of the utmost importance. This is particularly well understood by veterinarians engaged in work with dogs because the errors of feeding or care are often so very serious. But in poultry work the errors are apt to be even greater. With other species of animals correct husbandry is just as important but usually their care and feeding are better understood and are taken somewhat as a matter of course. Notwithstanding this we usually find either sick or healthy poultry treated with pills and bacterins when they need feed and care, or to be left alone. Some of the factors or conditions that influence poultry diseases are the weather, feeding, breed susceptibility, age resistance, and others. A study of certain diseases illustrates the importance of one or more of these factors while on the other hand other diseases are influenced very little or not at all. Certain diseases occur at certain times of the year and in this case the character of the weather is the determining factor, not the time of year. Weather that is advantageous for certain diseases usually occurs at certain seasons, hence they may be said to be seasonable. They occur during certain seasons or certain kinds of seasons and under certain kinds of climatic conditions. For instance, cholera

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

occurs in wet and cold weather. It is usually found most prevalent in the spring and fall, to some extent in the winter and much less in the summer. Cholera may occur during any rainy season but the usual Midwest summer is dry. Cholera is seldom seen in baby chicks, particularly when artificially brooded. This is not because chicks are resistant but rather because conditions are inimical. This characteristic of cholera suggests a method for its control, that is, sanitary measures should include provision for dry quarters and runs for affected flocks.

In contrast to cholera is fowl typhoid. Fowl typhoid is seen in the hot dry seasons. Usually it makes its appearance during the month of April, continues to affect more and more flocks during June and July, reaching its peak in August, declines rapidly in September and October, and amounts to little or nothing throughout the winter. As an aid to diagnosis, the seasonal distribution of disease is best exemplified in these two maladies—cholera and fowl typhoid. If one is without laboratory assistance and is in doubt as to which is affecting a flock, the time of year and the condition of the weather may help. In hot, dry weather one should favor typhoid, while in cold, damp weather a diagnosis of cholera would be most often correct.

Influences of Weather

Generally the respiratory diseases of mature birds are those of housing, dampness and cool or cold weather. Some of these, notably canker, may occur as a very benign form in dry, warm weather and in an extremely destructive form in cold, wet weather. Chickens and other poultry are not only liable to gross errors of feeding and management but are subject to a series of infections that are ever ready to infect and kill at any or all seasons of the year. In the spring, the newly hatched must be properly cared for if they are to survive. In chickens the first condition that is apt to affect them is pullorum disease. Danger of this is not past when they are apt to be affected with coccidiosis. These affections are overlapped in the summer with fowl typhoid, parasites and enteritis. Then in the fall, during cool, wet weather, the birds are most apt to be affected with cholera. At the same time or later, they are subject to canker, pox, tracheitis and the other respiratory diseases. No time of the year is free but rather the diseases overlap one another. Also nature, in her usual abundance, has supplied additional diseases or conditions that occur at any time of the year. Conspicuous among these are tuberculosis, fowl leukosis and parasitic infestations.

High Protein Feeding

The feeding of high levels of protein will, of itself, produce a disorder and loss, particularly in young birds. In general, it may be said that 15 per cent is too much meat scrap in baby chick mash. However, a 26 per cent protein ration is often fed to young growing turkeys. This does not cause losses but it is very close to the danger point and it does predispose to infections. The point is best illustrated by paratyphoid of turkeys. The first procedure in the control of paratyphoid is to place the young turkeys on a ration low in protein. The results obtained by such feeding, together with sanitary measures, are often phenomenal. Sanitary measures alone will often fail in such cases. The wise turkey-raiser knows that he is close to danger when feeding high levels of protein but contends, with justification, that he must get rapid gains if he is to realize a profit.

Breed resistance or susceptibility to disease is easily demonstrated in chickens. Also resistance is recognized in certain strains in the same breed. This, too, is an aid to diagnosis. For example, in individual cases we would suspect that heavy losses in New Hampshire Red chicks are due to pullorum disease, but that the same losses in White Leghorns are due to other causes. This is because the New Hampshire is notoriously susceptible to pullorum disease while the White Leghorn is resistant. The Mediterranean breeds are generally so resistant to pullorum disease that where it is found we are apt to think that they have been subjected to faulty management and have thus been made susceptible. This often proves to be the case. The other breeds stand intermediate between these extremes with many strains of the Rhode Islands more susceptible than others. Although the White Leghorn is resistant to pullorum disease, it is more susceptible to coccidiosis.

Fowl typhoid furnishes what is apparently a good example of increased resistance to disease in chickens. Only a few years ago, typhoid was very prevalent in the Midwest. Today it is exceptional to see a case. This seems to be due to an increased resistance of the chickens that are raised in the region. In other words, when any species of animals live in close association with a disease, some die of it, the remainder that are exposed are resistant, and they in turn produce young but are resistant. This is known certainly to be the case in fowl leukosis or range paralysis and forms one of the accepted methods for its control. Leukosis is especially destructive when introduced into a new region because of the large numbers of birds that are susceptible.

Once introduced into a locality or on a farm, it remains, so that chickens in such regions are repeatedly exposed and the offspring gradually become more resistant.

With certain qualifications too detailed to go into here, it may be said that it is good practice to send baby chicks from flocks that have had leukosis onto infected farms. The death loss will not be nearly so high as when chicks from free flocks are sent to infected regions. It is not uncommon to see a loss of 70 to 80 per cent where chicks free of leukosis are placed on infected farms. It is unfortunate that resistance to disease cannot be made perfect or 100 per cent by proper methods of breeding. It is also unfortunate that the causes or reasons for resistance are not all known. It is certain that it is partly genetic. Dr. Lambert was able to breed White Leghorn chickens that were 80 per cent resistant to fowl typhoid. But, after several years of effort, he could do no better. He concluded that there were factors which limited his work and that we do not yet know what these factors are. Clinically or epidemiologically this may be repeatedly observed. On the other hand, tuberculosis has almost certainly existed in chickens for a long time. Cholera was one of the first infectious diseases demonstrated, yet chickens are resistant to neither. The same is true of infectious enterohepatitis of turkeys. Surely resistance is not rapidly developed for some infections.

The Age Factor

Age resistance is of less importance. It may be that baby chicks are resistant to tracheitis of chickens, but in general it is true that a mature bird is more resistant than the immature; the more mature the more resistant. Again fowl typhoid is a good example. It often happens that fowl typhoid starts in the mature chickens where only a few sicken and die. It gradually works over into the young where the loss is very heavy. A history of an early mild loss in the mature chickens in contrast to a heavy later loss in the young is often sufficient for a diagnosis of fowl typhoid. Coccidiosis and infectious enterohepatitis are most severe in young birds.

Coccidiosis

Coccidiosis is caused by a number of protozoan parasites—coccidia. The symptoms and lesions vary slightly, depending on the particular species of parasite present. The symptoms are unthriftiness, diarrhea, ruffling of the feathers, and anemia. The appetite, the degree of emaciation and whether or not visible blood occurs in the feces depend largely on the type of coccidia

present and the severity of the disease. The lesions depend even more on the particular species of coccidia involved. One of the commonest and most severe forms is caused by *Eimeria tenella*, in which the characteristic bloody droppings are present. This is the usual spectacular type of the disease. The birds often take sick suddenly, pass bloody feces, have initial heavy losses and look so bad that one expects all to die. They may recover just as rapidly, so that the loss is not necessarily heavy. On the other hand, it may amount to 60 per cent or more.

A second type is due to infection with *Eimeria mitis*, in which lesions are confined to the upper portion of the small intestine. Lesions appear as small white areas about the size of a pin-point. In extreme cases the intestinal wall becomes thickened and the pin-point areas may coalesce into larger areas, reaching the size of a dime. There is unthriftiness and rapid emaciation. A third type, caused by *Eimeria acervulina*, also produces lesions in the anterior part of the small intestine and sometimes in the central or rarely in the posterior part. The chief lesions are small, whitish gray spots in the mucosa of the intestine. Hemorrhage is rare. The symptoms are unthriftiness, weakness, anemia and death. Still another type of coccidiosis is caused by the common *Eimeria maxima*, which inhabits the middle portion of the small intestine, where it causes a thickening with some hemorrhage accompanied by a thick mucus tinged with blood. In the cecal form symptoms usually occur four to five days after infection and the greatest loss occurs at that time. Birds that live seven or more days after infection usually recover.

In the other forms symptoms usually develop on about the sixth or seventh day after infection with losses on the eighth or ninth day. Again, birds that live three to four days after symptoms are noticed are apt to recover, although they may be unthrifty for a time. At the time symptoms are noticed, oöcysts are shed in the feces in large numbers. The typical case of coccidiosis in immature chickens is not difficult to diagnose, for it is characterized by an intense enteritis but without lesions elsewhere. Of course, the presence of free blood in the small intestine, and especially in the ceca, is diagnostic. Sometimes, and especially in older birds, the disease is apt to take a more benign form. The symptoms and lesions are not definite. A number of birds in an infected flock may be unthrifty, with perhaps some diarrhea and loss of weight and an occasional death. Enteritis is present but is not severe. It appears to be of a somewhat chronic nature. Other parasites may be present. It is such cases that are difficult to diagnose and at least a micro-

scopic examination of the intestinal content may be necessary to establish a diagnosis with certainty.

Fowl leukosis is very apt to cause the greatest loss in young chickens at about the time they are well feathered out. Chickens are also very apt to contract coccidiosis at this time. Thus we often see the two diseases in a flock at the same time. This happens so often that the question has arisen as to whether or not coccidia cause a paralysis. It often appears that leukosis is worse in such flocks and if such is true then one would conclude that the coccidiosis so weakened the chickens that the leukosis gained greater headway. The author is of the opinion, and with reason, that the two are distinct diseases, having no direct relationship whatsoever. For example, coccidiosis may cause a heavy loss in a flock without producing any paralysis and on the other hand leukosis may cause much paralysis and loss in a flock free from coccidia.

Renal coccidiosis of geese is so rare that it is of little or no significance. It is easily diagnosed from the symptoms and lesions. The main symptom is an emaciation that gradually becomes extreme. The lesions are confined to the kidneys. These organs are greatly enlarged, gray, and the tubules are gorged with urates and coccidia.

Pullorum Disease

Pullorum disease is one of the most important diseases of chickens. It also affects turkeys but to a less extent. It is so closely related to fowl typhoid that a consideration of one is not complete without a knowledge of the other. Indeed in many ways a knowledge of one is also a knowledge of the other. Pullorum disease is caused by *Salmonella pullorum* and fowl typhoid is caused by *Salmonella gallinarum*, two organisms so closely related that they are almost identical serologically and physiologically. Again, many of the lesions are almost, if not entirely, identical. The organs affected are the same. In chickens it has been shown experimentally that the fowl typhoid organism may remain alive in apparently recovered cases for many months and perhaps years. In such cases the location of the dormant infection is identical to that of *S. pullorum*, including the ovary, where it may produce lesions simulating pullorum infection. It must be said, however, that the ovary is not so often infected with the typhoid organism and that the organism has not been shown to be carried through the egg to infect the newly hatched chicks.

In chickens artificially infected with *S. gallinarum* there is much evidence to show that infected eggs might be laid because

the dormant infection may be found about the oviduct and in the ova. Apparently if such eggs are laid, they do not hatch, since of the thousands of dead baby chicks that are examined at various laboratories, the typhoid organism has not been found. Thus pullorum disease is an infection of all ages of chickens but it usually kills only young chicks, while fowl typhoid is a disease that kills at all ages but does not usually affect baby chicks because of the conditions under which they are kept. The last circumstance is fortunate from a standpoint of diagnosis, because if baby chicks were affected with both diseases it would be impossible to distinguish one from the other without a bacteriological examination. Sometimes chicks of six or eight weeks of age will die of pullorum infection and in such cases there is apt to be a question as to which disease is present. However, chicks that are affected with fowl typhoid are usually older than this and have usually had some association with mature chickens.

There is nothing pathognomonic about the symptoms displayed by chicks sick with pullorum disease. They are listless, stupid, sleepy and indifferent and huddle together. The feathers are ruffled and the wings droop. Diarrhea is often present but the affected chicks are usually not "pasted up behind." The respirations may be rapid, especially if the lungs are affected. They gradually become weaker. Losses run from a few chicks to 80 per cent. The symptoms are evident for an hour to a day or more. Most losses occur at from four to twelve days and reach their peak at about the tenth to twelfth day. Characteristic lesions occur in a large percentage of cases, although they may not occur in every chick that dies. For this reason it is often necessary to post a number of chicks from the same lot. On the other hand, only a few chicks may be dying of pullorum disease in any particular flock, whereas the greater loss is from some other cause.

The lesion of most significance is the whitish areas or nodules of leukocytic infiltration that occur in the pancreas, heart, gizzard, intestinal wall, liver and lung. The spleen also is enlarged. Such areas appear as small, light-colored nodules, usually pin-head in size but may be as large as a small pea. The nodules are apt to be larger in the involuntary muscles and in the pancreas. In involuntary muscles the nodules are distinct and circumscribed and take the shape and direction of the muscle fibers in which they lie. They are white in color and, near the surface of an organ, are elevated. In the pancreas and lungs the nodules are spherical and especially in the lungs they are surrounded by an area of acute inflammation. Very small nodules

are found throughout the liver and spleen as light-colored specks and are best seen just under the capsule. There is also a severe hepatitis characterized by enlargement, increased friability and a mottling in different shades of green and yellow. The egg-yolk is large and unabsorbed but this is seen also even in apparently healthy chicks. In general, these lesions are similar to those of typhoid, but typhoid usually affects older chickens and the lungs are not involved. Also typhoid is becoming rare.

Pullorum disease is a widespread disease of mature chickens and it is not exceptional to find flocks of heavy breeds that show 30 to 45 per cent reactors, although 20 per cent reactors is high for the light breeds. Adult fowls are often infected early in life but may be infected at any time. Usually no external symptoms are visible and the disease is revealed by the application of the agglutination test or by postmortem examination. In adult birds the infection is rather inactive, but an occasional bird may be lost and in some rare instances quite a large percentage of a flock may die. In the usual form the ovary is most often affected, although abscesses due to *S. pullorum* may be found anywhere in the body cavities and the liver is sometimes affected. The infection in the ovary produces easily recognizable changes in the larger ova. These are angular in outline, shrunken, partly resorbed or desiccated, and discolored dark brown or green. Rarely the content may be a dark fluid.

Whenever anything suddenly disrupts production in a laying hen, there will be a degeneration of some of the larger developing ova. Such degenerating ova must be differentiated from those infected with *S. pullorum*, a thing that is difficult to do in some cases. However, in pullorum disease more ova are often affected and they are found in all stages of degeneration. It is possible to raise baby chicks from pullorum-disease-infected hens, but such chicks are more difficult to raise than are clean chicks. Thus pullorum infection in baby chicks is usually a dormant or semidormant infection which requires something to give it a start and, once started, it causes a high mortality.

On the other hand, baby chicks are often so badly treated that they will die even though pullorum infection is not present. In this instance a little *S. pullorum* infection may also be present and postmortem of such chicks may show a little evidence of pullorum disease. In such cases a diagnosis of pullorum disease is incorrect. It too often happens that diagnosticians make the mistake of naming everything pullorum disease where a few lesions or organisms are found. The diagnosis of pullorum disease is not difficult, especially if the characteristic nodules are

present, but one should always use care to eliminate other causes for death loss.

Fowl Typhoid

Fowl typhoid is usually seen in flocks that have been previously healthy and at first only a few birds are affected. The disease is often slow in gaining a start, so that it may appear to be benign and of little importance for as long as two weeks. It gradually gains in severity until losses are extensive. It is apt to appear first in the mature chickens where it causes only moderate loss. From these it spreads into the young chickens where there may be a loss of as high as 75 per cent. Symptoms are a gradually progressing weakness, inactivity, jaundice, diarrhea, and elevation of temperature.

The lesions are severe catarrhal enteritis, jaundice, hepatitis and emaciation in protracted cases. The enlargement of the spleen is pathognomonic in this type of disease. The spleen is enlarged in all acute cases and the liver enlarged or changed in color in almost all cases. The enlarged, soft, easily broken and torn liver is mottled with various colors, mostly shades of brown and yellow or green (bronze), although in some cases the organ is a uniform brown or green. From the cut or torn surface of the liver the blood drips, thin, watery and yellowish or dirty looking. White circumscribed discrete nodules are often found in the myocardium. The enteritis has no characteristic features. Acute congestion of the intestine is absent. The lesions in the liver are to be differentiated from liver lesions seen in acute cholera. In cholera, parts of the liver only may appear to be chiefly affected, the liver may appear hard and firm as if cooked, lighter in color and with the discolorations in streaks of large areas, whereas in typhoid the liver is soft and uniformly affected throughout.

Fowl Cholera

Fowl cholera, a wet and cold weather disease, occurs in a septicemic form and a localized form or in a combination of the two. The localized form usually terminates in a septicemia and in flocks where the sick birds are mainly affected with some localized form, such as oedema of the wattles, individual cases of septicemia also will be seen. This circumstance is often an aid to diagnosis. As an example, one may examine a flock in which there is a roup or cold-like disease and may be uncertain as to its cause. A history that a few birds have also died of an acute septicemic disease is good evidence that the condition is caused by *Pasteurella avicida*. The rapidity with which a

flock of chickens dies of cholera has been overemphasized. Ten to 70 per cent or more of a flock may die in two or three hours in case of poisoning but this is almost never the case with cholera. When a large number of susceptible chickens eat the flesh of one that had died of cholera, then these may sicken at nearly the same time and many will die within 24 to 36 hours. However, this is unusual. The disease spreads more slowly.

In the generalized or septicemic form the symptoms are those of an acute septicemia. The birds are visibly sick for an hour or so to one or two days. The gross lesions are largely confined to the liver, serous surfaces, the peritoneal cavity and the duodenum. In the liver there is an acute parenchymatous inflammation. The organ is usually light-colored, rarely green, firm, and often contains light-colored, firm areas and streaks much like cooked liver. The serous surfaces show petechiae, particularly over the fat of the abdomen. An animal that dies in a struggle is particularly apt to show small petechiae of the heart. These hemorrhages are caused at the time of death and are of no diagnostic value except to show how the animal died. This is particularly true of chickens, so that petechiae of the heart are of little diagnostic value. On the other hand, this is not true of cholera in turkeys and geese—the petechiae are larger, are produced by the disease itself. They are usually present and are diagnostic. The abdominal cavity may contain a mass of cheesy exudate, similar to light-colored, boiled egg-yolk. Acute inflammation with congestion is present in the anterior part of the small intestine.

The cholera organism may localize in many parts of the body. A cold or roup-like form has been mentioned. In this the infection may involve the bones of the head, especially about the ears and at the base of the brain. In this instance a yellow, cheesy exudate will be found within the bones. The wattles may be infected. In this case the wattles become rapidly swollen, red and hot without apparent cause. It is usually seen in males with large wattles. The infection may also localize in the tendon sheaths, particularly of the legs. The chicken is lame. The joints become hot, painful and swollen. In the Middle West, cholera or goose septicemia is still the most important infectious disease of geese. It is common in turkeys and rare in ducks.

Tuberculosis

Tuberculosis is one of the major diseases of poultry, especially of those species that are not web-footed. The symptoms and lesions are recognized everywhere so that it is rare that a case

of tuberculosis is not properly diagnosed. However, there are some diseases that might at times be mistaken for tuberculosis. Any disease that produces spherical lesions of the internal organs might be so mistaken. As examples, we have fowl leukosis and infectious enterohepatitis in some instances. Fowl leukosis may produce nodules in the liver and spleen that simulate tubercles. Some cases of enterohepatitis also show lesions that resemble tubercles, that is, the nodules on or near the surface of the liver are rounded, white, and rather firm. In this case the rather unnatural lesions may be partly due to bacterial infections that have also affected the organ along with *Histomonas meleagridis*. A macroscopic examination of the cut surface of such lesions usually differentiates them from tuberculosis. In tuberculosis the larger tubercles show a tough capsule and its content is caseated or necrotic, whereas other nodular lesions either lack the tough capsule or the necrotic center or both.

PARATYPHOID OF TURKEYS

Paratyphoid of turkeys is one of the important infectious diseases, especially of young poults that are being forced for rapid gains. The symptoms vary somewhat, depending on the length of time individuals are sick. Death is sudden in many cases. Birds may sicken and die in twelve hours or less. Other cases linger for two days or more. In these cases there is weakness, the feathers are ruffed, the wings drag, there are slow, unsteady movements and a watery diarrhea. The poults stand with the eyes closed and the head under the wing or hanging forward. The disease also occurs in older birds but does not cause such high death losses. The lesions are confined to the ceca, liver, kidneys and intestine. The ceca are usually distended with great masses of cellular débris and exudate, which is firm and cheesy. The small intestine is highly inflamed. Hepatitis and nephritis are usually seen. This is evidenced by swelling and congestion. Absence of necrotic areas in the liver differentiates paratyphoid from blackhead. It occurs as a rather benign disease in chickens. In ducks it is known as "keep disease."

DISCUSSION

DR. F. R. BEAUDETTE: I should like to take exception to Dr. McNutt's statement to the effect that fowl typhoid is not found in baby chicks. In our experience, we have isolated the organism from baby chicks even under a week old, on many occasions. As a matter of fact, we have had more outbreaks in young chicks than in adult birds. I believe the infection is transmitted through the egg occasionally, in the same manner as it is transmitted in pullorum disease.

We had the experience of finding numerous outbreaks of fowl typhoid in baby chicks which originated from a certain hatchery. Some years

ago, I obtained hatching eggs from this hatchery, and incubated them. From incubated eggs I isolated the fowl typhoid organism. I have also isolated it from the ovaries of birds on several occasions. This, however, as pointed out by Dr. McNutt, may not be evidence of transmission through the egg—that is, the mere finding of infection in the ovary—but we have found it in baby chicks on many occasions, which have had no other contact with the infection.

Pioneer Practitioner Honored

Dr. J. C. Whitney, of Hillsdale, Mich., who served as president of the Hillsdale County Fair Association for the year 1936-37, was honored by his associates at the completion of his term of office. The members of the Whitney Veterinary Club were the guests of the Fair Association at the races held on September 29. In the evening a dinner was given in honor of Dr. Whitney, attended by more than 50 of his friends. This pioneer Michigan practitioner was graduated from the Ontario Veterinary College in 1883.

Chappel Award Nominations to Close November 15

Following its established custom, the Chappel Foundation will close its nomination period on November 15, 1937, for the 1938 winner of its coveted award. According to W. E. Armstrong, director of the Foundation, the list of nominations this year has again shown an increase in interest on the part of all dogdom in the Chappel Award. All nominations are to be addressed to Chappel Kennel Foundation, Rockford, Illinois.

Phi Zeta Smoker at Cornell

Alpha chapter of Phi Zeta, honorary society in veterinary medicine, held a smoker in Willard Straight Hall, the student union building at Cornell University, the evening of October 19, 1937. The meeting was open to all veterinary students and members of the faculty. Dr. E. R. Day, Cornell's newly elected president, presented a loving cup to Mr. Theodore Allen, highest ranking member of the freshman class for the year 1936-37.

Prof. A. L. Romanoff also presented some very interesting motion-pictures in color, showing the development of the chick embryo. The meeting served as a get-together of students and faculty, as well as to do honor to scholarship in the freshman class and to make the undergraduates acquainted with the aims and purposes of the Society. H. L. G.

EARLY RESEARCH AND PRESENT-DAY PROBLEMS IN HOG CHOLERA IMMUNIZATION*

By HENRY HELL, *Wilton Junction, Iowa*

In compiling the necessary material for this discussion, there has been no attempt made toward compilation of minute and exact historical or statistical data, but to discuss briefly deductions and conclusions drawn from research and experimentation in both laboratory and field. It is the purpose of this paper to discuss all problems from the angle of the practitioner and to call special attention to the problems that materially interfere with success in practice. Pursuing this discussion, we must be ever alert against making deductions or drawing conclusions from speculative reckonings that may not have foundation in fact.

The motive for the compilation of material for this discussion is the hope that some little additional light may be shed on one or more of the vexing conditions that arise from time to time in hog practice. Every Corn Belt practitioner, at some time or another, has come face to face with some condition that completely upset his expectations of results in a herd of serum-virus-treated hogs. You are all familiar with the picture. You vaccinate five or ten herds of shoats with the same serial of both serum and virus. All do well except one herd, which goes completely "haywire" from six to ten days after treatment. Then you hear that old familiar howl, "Poor serum."

We firmly believe that successful immunization with serum and virus is sometimes materially interfered with by the presence of certain bacterial infections, ordinarily referred to as secondary invaders, present in the herd at the time serum and virus are administered.

Every effort should be made by the practitioner to recognize the presence or absence of the secondary infections before the administration of serum and virus. All responsibility for results obtained in apparently healthy herds of shoats, after serum and virus treatment, rests squarely on the shoulders of the practitioner. To ruin a herd of hogs with poor vaccination results is, in the eyes of the average herd-owner, an unpardonable sin.

We must not lose sight of the fact that the treatment of 100 shoats with serum and virus is a major operation, involving a monetary consideration above that of average routine, and there-

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

fore calls for our best judgment. We know in a general way that herds with pen infections or heavy parasitic infestations are poor vaccination subjects. We find also that herds, apparently holding their own, with no outward indication of these infections, "go wrong" after vaccination.

A fact worthy of consideration is that we produce a decided leukopenia with the serum and virus treatment. Are we justified in assuming that the defensive forces of the body (phagocytosis) have been lowered to an extent that an infection, normally held in check by the defenses of the body under this condition, gains the upper hand?

Increased dosage of serum for these borderline cases is widely practiced and is a commendable procedure—if you do not ride a good horse to death. Herds definitely infected with secondary invaders should not be treated with serum and virus unless surrounding conditions urgently demand it. If possible, these herds should be placed on a correct diet, clean ground, and medicinal treatment of the alkaline variety.

In our early vaccination difficulties we carried along with us too many recollections of "wild cat" serum production in 1913. We were content, when trouble appeared, to agree with the owner that it must have been a batch of poor serum and passed the buck to the producer. Now, with the production of a fairly uniform quality of serum by all reputable producers, it becomes necessary for the practitioner to shoulder the major portion of the responsibility for vaccination results.

Hog Cholera and Swine Plague

In 1885, Dr. D. E. Salmon and Dr. Theobald Smith isolated two organisms that we now know as *Salmonella suipestifer* and *Pasteurella suiseptica*. These early investigators were firm in their opinion that these two organisms were the specific causative factors of hog cholera and swine plague, reckoned then as two separate and distinct diseases.

From 1885 to 1905 (when the filtrable virus was demonstrated by Drs. A. E. de Schweinitz, M. Dorset, B. M. Bolton and C. N. McBryde), considerable animal experimentation was carried on by Salmon, Smith and others, with both organisms. Pure culture inoculations (probably contaminated with filtrable virus) invariably resulted in the recovery of both organisms from the inoculated animal. This is in accord with our present-day finding of both *S. suipestifer* and *P. suiseptica*, of varying degrees of pathogenicity, in hogs with or without hog cholera.

For 20 years immediately following Dorset's demonstration of the filtrable virus, very little thought was given these organisms of Salmon and Smith. It seems, however, that they are again on our doorstep and ask for consideration. The evidence presented by laboratory and field makes it necessary that we consider *S. suipestifer* infection as a major factor in the difficulties appearing any time six to ten days after serum and virus treatment. *P. suiseptica*, from what we are able to learn from commercial laboratories, is not of high pathogenicity in this Corn Belt region.

Pasteurella Suiseptica

Let us remember that this organism belongs to the hemorrhagic septicemia group and has a wide distribution and a wide range of pathogenicity, ranging from the most harmless saprophyte to the buffalo strain, capable of producing death in the hog in about 48 hours.

In considering this organism, its pathogenicity becomes our objective study, and as most private laboratories report low pathogenicity for this section, it would seem justifiable to regard this organism of secondary importance to the more consistently virulent *S. suipestifer*.

Salmonella Suipestifer

Text-books classify *S. suipestifer* as belonging to the intermediate or Gaertner group, which appears to be between the typhoid and colon groups. Some seven or eight organisms have been isolated from this group. All seem to be quite uniformly virulent and quite closely related. Clinically they may present local or septicemic syndromes.

Research workers furnish us with the following information. Murray and associates found *S. suipestifer* to be primarily a local intestinal infection. McBryde found *S. suipestifer* in pure culture in the mesenteric lymph-nodes and lung tissue. McBryde also found the organisms in the heart-blood, spleen and kidney, all indicating that the organism circulates freely and may be viewed as being of the septicemic type. Clinical interpretations of these findings would lead us to believe that the suipestifer organism was taken up from the intestinal walls by the lymphatics in their function of fat assimilation, carried to the mesenteric lymph-nodes, then to the great lymphatic (thoracic) duct, then to the anterior vena cava and then into the pulmonary circulation. This may explain why a pure culture of *S. suipestifer* may be found in the lung tissue. Finding the organism in the spleen or kidney would indicate that it gained entrance to

the general circulation. Is it then not quite logical to conclude, from the clinical point of view, that a septicemic form of suipestifer infection does exist?

Clinically we meet up with a condition, either in susceptible hogs or in hogs that have been vaccinated and are supposed to be immune, that presents many of the symptoms of hog cholera. Still there is something lacking to give a clear picture of a cholera herd. Temperatures run up to 108° F., there is lung involvement, sometimes profuse petechiation, enlarged and discolored lymph-nodes, and the whole layout presents about the picture of yard type of hog cholera.

Bacterin Treatment

When you meet this condition in practice and are sufficiently sure of your diagnosis that it is not cholera, treat the herd with a generous dose of bacterin consisting of entirely chemically devitalized broth cultures of *S. suipestifer*. Put the hogs on a light diet with a laxative, alkaline medication.

Clinical experience does not cover enough ground to warrant drawing definite conclusions, although I have seen herds make almost a complete recovery within five days, in both immune and susceptible herds. The susceptible herds received no serum-virus treatment.

Evidence seems to warrant the conclusion that suipestifer infection does exist in the septicemic form and will respond to bacterin treatment. Here it seems we must again take up the work of Salmon and Smith. We must again deal with these bacterial invaders, not as specific factors causing hog cholera, but as contributory factors that so materially interfere with successful serum and virus treatment.

Local enteric infection, as found by Murray and associates, may be treated with bacterin, although this form responds to medicinal treatment.

Early Breaks

Breaks occurring within ten days after serum and virus treatment may be attributed to bacterial infections rendering the animals more susceptible to virus, or shall we put the shoe on the other foot and say that the administration of virus weakens the leukocytic defenses of the animal against bacterial attack? Whatever view you take, the combination of virus and bacterial invasion does not work out so well.

Susceptibility to virus varies greatly in different litters of pigs. This was clearly shown in the experiments of McBryde, to determine the smallest dose of serum necessary to protect

against 2 cc of virulent virus. It was found that some litters were fully protected by 6 cc of serum against 2 cc of virus for a 35-pound pig, while other litters of the same weight showed no protection at all from the same dose of serum.

It is therefore up to each practitioner to work out for himself a dose table suitable to the conditions that prevail in his locality. Sufficiently large doses of serum are of prime importance in avoiding these so-called early breaks. Do not underestimate the weight of the pig. The rough, poorly nourished pig should receive relatively larger doses than the smooth, vigorous individual.

Late Breaks

When we set out to consider late breaks we should carry two things in our minds: (1) recurrent hog cholera and (2) factors other than hog cholera. I like to think of some of these breaks that are definitely hog cholera as recurrent from the virus infection at the time of serum and virus treatment. It has not been definitely established in this country just how long after vaccination virus is retained in the body of the hog. These late breaks occur mostly from four to eight weeks after vaccination. The uniformity of the time of their occurrence would preclude against outside infection that might occur anytime after the hog had lost its passive serum immunity.

In considering breaks not definitely hog cholera, the acute septicemic form of suipestifer infections will give you a line of antemortem and postmortem findings that are hard to differentiate from cholera.

If the herd has been serum-virus-treated, the record of the products used is a great help. If you are sure that the virus used was fresh and potent in doses of 2 cc or more and had not been abused by exposure to heat and light, I would feel safe in excluding hog cholera from the diagnosis.

I do not recall revaccinating a herd of hogs in my own practice since 1915 (when all simultaneous virus carried a full 1 per cent of phenol as a preservative). I am definitely opposed to revaccinating hogs that have received 2 cc or more of a good reliable virus when the pigs were at least four weeks old and thrifty at the time of vaccination. There seems to be virus on the market that is of low potency.

In a neighboring state, where lay vaccination is encouraged by the Farm Bureau, many late breaks have occurred. No doubt a large percentage of these are recurrent hog cholera. By this we mean that virus has been retained beyond the passive serum

immunity and become active. In these cases serum and virus in large doses is the only rational treatment.

Serum and Virus Versus Vaccines

Many of us who have traveled along the serum and virus route for more than a quarter of a century, followed it through its swaddling clothes, its growing pains, its adolescent stage, into full maturity as a near-perfect immunization procedure, are firmly of the opinion that further proficiencies can and will be made in the application of this mode of immunization to make it within a small fraction of 100 per cent perfect.

We have at our disposal the conferring of a solid, life-long immunity if we will avail ourselves of the necessary material and procedure. The requisite factors are a herd of pigs that are reasonably healthy and thrifty, the youngest not less than four weeks old; not less than 2 cc of fresh, highly virulent virus, with sufficient serum to protect against the virus under all circumstances; and not less than 25 cc of serum for any pig, no matter how small. If the pig is too small to require this amount of serum, the chances are it is too young to develop a solid immunity.

Tissue vaccines such as the crystal violet, formolized or otherwise chemically attenuated virus, are still very much in the experimental stage. Conclusions drawn from experimentation reported thus far would lead one to believe that the immunity conferred did not withstand virus inoculation at a later date in about 30 per cent of the animals so inoculated.

Our goal has always been to develop a 100 per cent, solid, life-long immunity. If we will bear in mind the requisite factors necessary and not allow ourselves to deviate from them, we will have accomplished our objective with the serum and virus treatment.

Correspondence from a neighboring state indicates that *S. suipestifer* contamination of simultaneous virus is still a factor in serious losses following serum and virus treatment. Cases are cited in sufficient numbers to substantiate this contention.

It is well known that many producers check their simultaneous virus against this and other contaminations while others may not do so.

Practitioners will welcome, from research and regulatory forces, additional information on suipestifer infection as a complication of virus infection. We would like to see excluded from interstate commerce all simultaneous virus that has not been

checked for bacterial contamination and is not up the highest peak of virulence.

We would like to see 2 cc the minimum dosage recommended on all virus labels, and 25 cc the minimum dosage on all serum labels, in view of the fact that it has been proclaimed and has not been contradicted in open meeting of either this Association or the United States Live Stock Sanitary Association, that 65 per cent of the hog cholera outbreaks are the result of injudicious use of virus.

It is therefore timely to call attention again to the lack of supervision of the use of virus in the field by allowing the ignorant and the careless to handle a death-dealing virus with little or no restriction. Lay vaccination and the carelessness of a few within our own ranks constitute the greatest menace to the hog industry of the Corn Belt.

B. A. I. Veterinarians Publish Directory

Under date of July 15, 1937, the National Association of Bureau of Animal Industry Veterinarians published a new membership directory. This was the first edition to be published since March 15, 1933.

The new edition lists 1,020 members, as against 1,137 members listed in the 1933 edition. An analysis of the 1937 membership shows that 539 (52.8%) of the B. A. I. veterinarians are members of the American Veterinary Medical Association. This compares with 571 (50.2%) B. A. I. veterinarians who were members of the A. V. M. A. in 1933.

Concerning Eligibility for Attendance at the Conference of Official Research Workers in Animal Diseases of North America

The next annual meeting of the Conference of Official Research Workers in Animal Diseases of North America will be held at Hotel Sherman, Chicago, Tuesday, November 30. In order that eligibility for attendance at this meeting may be clearly understood, it seems advisable to quote Article II of the Constitution and By-laws of the organization. Article II reads:

> Research workers regularly employed by the United States government, or by any national, state or provincial government in North America and charged with official investigation of animal diseases or products used in their control shall alone be eligible, ex officio to active membership in the Conference.

WILLIAM H. FELDMAN, *Secretary-Treasurer.*

INTRAVENOUS USE OF SODIUM IODIDE IN ACTINOMYCOSIS*

By JAMES FARQUHARSON, *Fort Collins, Colo.*
Colorado State College

Actinomycosis is one of the most prevalent conditions encountered in cattle practice in the West. We see hundreds of cases annually and the economic loss from this condition is inestimable.

In discussing treatment of actinomycosis, the word "actinomycosis" will be used in the broadest clinical sense of the term. Our newer knowledge of what has been regarded as actinomycosis as an entity now also embodies actinobacillosis. Generally speaking, actinomycosis causes more or less of a chronic, suppurative osteomyelitis, resulting in destruction and rarefaction of bone; whereas, the infection of soft tissues, such as the lymph-glands, skin, vulva and mammae is generally caused by actinobacillosis.

The discouraging results in treatment, particularly in cases in which bone is primarily affected, are too well known and accepted. Bone lesions in the past have usually resisted treatment, either surgically or from the administration of iodides *per os*, or a combination of the two. Many cases in which soft tissues alone are affected are either inaccessible to surgical treatment, or many are not entirely corrected by the administration of iodides. A review of authentic literature shows that between 50 and 70 per cent of so-called actinomycosis is arrested or reacts favorably to the administration of potassium iodide by the mouth.

Quoting Mohler and Shahan[1]:

> The treatment of lumpy or big jaw is almost entirely surgical if the bone itself is involved. Disease of the jawbones is at best a very serious matter, and treatment is likely to be of no avail, particularly if not begun early.

They further state:

> Subsequent accumulated evidence indicates that the disease of the soft tissues, which is usually actinobacillosis, will generally yield to administration of iodine in one form or another, but that actinomycosis may not be materially improved by that chemical.

Without question this has been the consensus of all the profession engaged in practice. The use of potassium iodide in the treatment of actinomycosis and actinobacillosis necessitates the

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

animal being placed on this drug twice daily for a period of seven to ten days until iodism appears, then a week or ten days of rest and a similar treatment repeated. This entails considerable cost, and one must depend upon the owner to give it, which in many instances is neglected. In the animal receiving such treatment there is considerable loss of flesh and a heavy loss in milk production in lactating cows.

Surgical treatment either by extirpation of the infected tissue, or by incision and drainage, or by the injection of some iodine preparation into the lesion, is not at all satisfactory because of the slowness of healing. It means repeated daily treatment, formation of permanent fibrous enlargements, and a tendency for the condition to recur.

A review of the literature reveals that potassium salts injected intravenously are dangerous. Holland[2] reports that the potassium ion in the blood, in excess to the physiological need, is toxic. Keith and Binger[3] state:

> Potassium salts when injected intravenously into animals can cause rapid toxic effect, particularly on the heart.

Mathison[4] believes that the toxic action of the intravenous injection is due to the high concentration of potassium that immediately reaches the heart muscle. Amberg and Helmholz[5] found that the lethal dose of potassium chloride and sulfate was very small when injected intravenously in the guinea pig. The experiments of Keith and Binger[3] lead them to the following conclusions:

> We believe that the results of experimental studies on animals contraindicate, at present at least, the intravenous administration of any considerable amount of potassium salts in clinical medicine. On the other hand we have shown that normal individuals can take a large dose of a potassium salt by mouth without demonstrable toxic effects.

Sodium salts are much less toxic and can be tolerated far beyond the physiological need of the body. With these things taken into consideration, and the fact that the bovine as a species is quite susceptible to the action of iodine and with the combined toxic action of potassium, it was deemed safest to use sodium iodide intravenously.

For the past four or five years, we have been using sodium iodide intravenously in the treatment of actinomycosis and actinobacillosis. After this long trial we can report that it is much superior to any of the older methods of treatment.

The dosage is gauged according to the weight. Animals weighing 1,000 pounds are given one ounce of sodium iodide dissolved in one pint of sterile distilled water. Those weighing 1,500

pounds are given 1½ ounces in the same amount of water. Those weighing approximately 500 pounds should be given four to six drams of sodium iodide. Young animals seem to tolerate relatively larger doses. The dosage for animals between these weights should be proportionably the same.

The method of administration is with any intravenous outfit, given into the jugular vein, using a 16- or 18-gauge needle. It should be given slowly and care must be exercised, particularly toward the termination of the injection, to watch for a systemic reaction. This is manifested by slightly increased restlessness, increased respiration and heart action, occasionally lacrimation, and apparent vertigo. If this appears, further administration should be stopped immediately. Probably less than five per cent of animals so treated will show such a reaction. There are no unfavorable results if such a reaction does occur, and no deaths have been experienced from its use. Animals debilitated from extensive lesions withstand treatment equally as well as strong individuals.

Animals treated in this manner do not show symptoms of iodism. A very small number so treated may show slight impairment of appetite for about two days. Milk cows may show a very slight decrease in lactation for about the same period of time. It will cause a high percentage of abortions in pregnant animals. Therefore, the owner should be warned of this, otherwise treatment should be delayed until the cow has calved. No surgical treatment is necessary as an adjunct to the intravenous use of sodium iodide in the treatment of lesions involving soft tissues. In cases where abscesses are so large as to cause impaired function mechanically, then it is always best to incise and drain along with the intravenous medication. The decrease in the size of the soft tissues involved is much more rapid and certain than with the use of potassium iodide. Snoring due to deep seated pharyngeal abscesses, which is usually accompanied by pronounced dyspnea, shows rapid amelioration for two or three days when symptoms disappear. "Woody tongue" is relieved in twenty-four hours so that the animal can eat and drink. There is a much higher percentage of permanent cures from the intravenous use of sodium iodide than from other methods or the administration of potassium iodide. One treatment is usually sufficient in soft tissue lesions.

Bone lesions should be treated surgically by trephining or any other convenient method and the soft bone removed with a curette. This is accompanied by the intravenous use of sodium iodide. Invariably the process is arrested and a remarkable dim-

inuation in the size of the bone will result. A second intravenous injection is recommended in ten to 14 days.

Summary and Conclusions

Sodium iodide administered intravenously has been successfully used in the treatment of actinomycosis for a period of over four years.

Advantages of the intravenous use of sodium iodide in treating actinomycosis are: Treatment of this condition is too often taken care of by the layman through his knowledge of the use of potassium iodide. Laymen, after seeing the advantages of the use of sodium iodide in comparison with that of potassium iodide, are forced to employ a veterinarian because of its method of administration.

Sodium iodide treatment does not cause a loss of flesh in animals that are being fitted for beef; any slight decrease in lactation in dairy cows is but temporary; animals in which actinomycosis appears previous to shipment can be treated without any bad results or delay in shipment.

The intravenous administration of sodium iodide is productive of a higher percentage of recoveries and more certain results than with any other form of treatment; it is safe to use; only one treatment is necessary; the cost is negligible; iodism is not produced, and bone lesions react favorably to its use.

The intravenous use of sodium iodide usually causes abortion in pregnant animals.

In addition to the method of medicinal treatment, it returns to the profession a service that can be rendered only by a veterinarian.

Acknowledgment

I desire to express my appreciation to Dr. N. J. Miller, of Eaton, Colorado. Both of us started working on this at about the same time and Dr. Miller has coöperated in helping to make it successful.

References

[1]Mohler, J. R., and Shahan, M. S.: U. S. Dept. Agr. Circ. 438 (1937).
[2]Holland, J. W.: Medical Chemistry and Toxicology, p. 214.
[3]Keith, N. M., and Binger, M. W.: Jour. Amer. Med. Asso., cv (1935), pp. 1584-1590.
[4]Mathison, G. C.: The effect of potassium salts upon the circulation and their action on plain muscle. Jour. Physiol., xlii (1911), pp. 471-494.
[5]Amberg, S., and Helmholz, H. F.: The detoxifying action of sodium salts upon potassium salts on intravenous injection. Jour. Phar. & Exp. Therap., viii (1916), p. 120.

The International Live Stock Exposition will be held in Chicago, November 27-December 4.

THE EFFECTS OF VARIOUS SODIUM, POTASSIUM, AND CALCIUM SALTS ON RESPIRATION AND BLOOD-PRESSURE IN THE DOG*

By E. A. HEWITT, D. A. GREENWOOD *and* V. E. NELSON
Laboratories of Veterinary Physiology and Physiological Chemistry, Iowa State College, Ames, Iowa

It has long been known that calcium, sodium and potassium ions in the blood and lymph are necessary for the proper functioning of certain body tissues. The compounds used in these studies were selected because of their importance from a clinical and metabolic standpoint. The experiments recorded in this paper were performed in conjunction with a series of studies on the effects of various fluorides and related compounds upon respiration and blood-pressure, the results of which have been or are in the process of being published.

DESCRIPTION OF THE ANIMALS

All of the experiments were performed on dogs. Seven dogs were used, four males and three females. The smallest weighed 5.49 kilos and the largest 20 kilos. The exact ages of the dogs were not known, although they were all mature animals.

METHODS AND APPARATUS

The experimental methods are those ordinarily used in pharmacological and physiological studies involving the use of a long-paper kymograph, mercury manometer, pneumograph, time marker and signal magnet. Three per cent pento-barbital-sodium (Abbott), administered either intraperitoneally or intrapleurally on the basis of 30 mg per kilo of body weight, is ordinarily employed for anesthesia. The general procedure in performing an experiment was to inject small quantities of the compound, after a preliminary tracing had been made. The preliminary tracing was assumed to be normal for the animal under anesthesia; thus the effects on the blood-pressure and respiration resulting from the injection of the salts could be evaluated. The dosage of the salt was increased gradually, until a significant change was produced on respiration and blood-pressure.

SODIUM CHLORIDE

The toxicity of sodium chloride is of special interest, due to the fact that this salt constitutes the greatest amount of salt

*Received for publication, October 14, 1936.

present in the plasma and is largely responsible for the osmotic pressure exerted by this fluid, and also because sodium chloride plays such a vital rôle in the metabolism of the tissues.

Table I records the results obtained on respiration and blood pressure as a result of the intravenous injection of a 30 per cent solution of sodium chloride into a male dog weighing 18.18 kilos. It will be noted that the initial injection of 300 mg of sodium chloride produced an elevation of the blood-pressure from 160 to 170 mm of mercury. The respirations remained unchanged. A second injection of like amount again caused an increase in blood-pressure from 166 to 170 mm of Hg. After one minute,

TABLE *I—Effect of sodium chloride (30 per cent solution) on respiration and blood-pressure (male dog, 18.18 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)
2.50	150	20	
2:59	160	22	0.300
2:59:30	170	22	
3:00	164	23	
3:01	166	23	0.300
3:02	170	22	1.500
3:02:15	178	22	
3:02:30	150	22	
3:02:45	180	25	
3:03	176	25	3.000
3:03:30	120	50	
3:04	186	26	
3:05	184	25	3.000
3:05:15	160	51	
3:05:30	180	51	
3:05:45	120	50	
3:06	184	50	
3:07	184	50	3.000
3:07:30	160	56	
3:09	180	50	4.500
3:09:30	160	56	
3:10	180	56	6.000
3:12	170	56	9.000
3:13	140	56	
3:14	172	50	15.000
3:15	124	42	
3:16	146	56	15.000
3:18	96	50	
3:19	100	Ceased	
3:19:30*	60	Ceased	
3:22†	30	Ceased	
		Total	60.600

*Heart-beat strong but slow.
†Heart-beat ceased.

the injection of 1.5 gm of the salt caused a further increase of blood-pressure to 178 mm of Hg. A two-minute interval was allowed to elapse, during which the blood-pressure fell to 150 but rose immediately to 180 mm of Hg, dropping back in a short time to 176. During this time the respirations were not significantly affected, although there is noted a gradual increase from 20 per minute to 25 per minute. Subsequent injections, however, produced marked effects upon the respiratory rate. The next injection of 3 gm of salt caused a 100 per cent increase in the number of respirations per minute, although the rate quickly dropped back to the previous rate of 25 per minute. The immediate effect of this injection upon blood-pressure was to cause it to fall to 120 mm of Hg, but it quickly returned to the level at the time of injection, namely, 184 mm of Hg. Subsequent injections had a marked effect upon respiration, causing it to be maintained at a high rate until the lethal effect was produced and caused some fluctuation in the blood-pressure, which in general was maintained at a relatively high point as compared with the preliminary level. The lethal dose of sodium chloride for this animal was 60.6 gm. Autopsy revealed edema and hemorrhage of the lungs and liver.

The results observed in this experiment can perhaps be explained by assuming that there are at least three ways in which the salt exerts its effect:

1. The osmotic pressure effect, resulting from the introduction of the hypertonic solution into the blood at first caused a withdrawal of fluid from the tissues into the blood-stream, which thereby increased the blood-volume and thus the blood-pressure. Later, the direction of fluid passage was from the vessels to the tissues.

2. There was a direct effect upon the medullary centers, which was probably an ionic effect. The general effect was to stimulate the vasomotor and respiratory centers until the lethal dosage was reached, when a collapse or paralysis of those centers occurred.

3. The third probable effect was one of cell permeability. The capillary cells became more permeable and allowed fluid to escape into the tissue spaces, which tended to cause a concentration of the blood and thereby a decrease in volume with a fall in blood-pressure. However, the stimulating effect on the vasomotor center was able to maintain the blood-pressure at relatively high levels in spite of the passage of fluid into the tissue spaces. This increased permeability of the capillaries was indicated by the edema and hemorrhage in the lungs and liver.

SODIUM CITRATE

The toxicity of sodium citrate is of interest, because sodium citrate is frequently used in blood transfusions as an anticoagulant and is perhaps the most popular anticoagulant for this purpose in use today. Petersen, Hewitt, Boyd and Brown[1] were able to produce a condition of hypocalcemia in cows, which closely simulated parturient paresis, by the intravenous administration of 20 per cent solution of sodium citrate. This condition could

TABLE II—*Effect of sodium citrate (20 per cent solution) on respiration and blood-pressure (male dog, 5.49 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MIN.)	AMOUNT INJECTED (GM)	PULSE PRESSURE (MM)	REMARKS
3:39	132	28		2	
3:47	135	28	0.20	2	
3:47:15	103			2	R. Temporarily ceased
3:48	126	35		2	
3:49	132	28	0.10	2	
3:50:15	122	40		2	
3:50:30	128	30		2	
3:51	130	30	0.20	2	
3:51:15	112	50		4	
3:51:30	123	28		2	
3:53	123	63			R. Decreased in amplitude
3:54	130	49	0.30	2	
3:54:15	100			2	R. Temporarily ceased
3:55	128	42		2	Gasping and dyspnea, tetany
3:57	124	70		2	Tetany ceased
3:58	126	56	0.20	2	
3:58:15	103			2	R. Temporarily suspended. Tetany
3:59	122	70		2	R. Shallow, dyspnea
4:00	122	66	0.20	2	R. Increased in amplitude. Dyspnea decreased
4:00:15	82			2	R. Temporarily ceased
4:01	120	50		2	R. Shallow, irregular. Dyspnea intervals suspended
4:03	122	40	0.40	2	R. Regular and increased amplitude
4:03:30	52				Heart weak. R. suspended
4:04	110	28			R. Irregular. Dyspnea
4:05	120	25	0.40		R. Irregular. Dyspnea
4:05:15	40				R. Suspended. Heart feeble. Death
		Total	2.00		

R. = respirations.

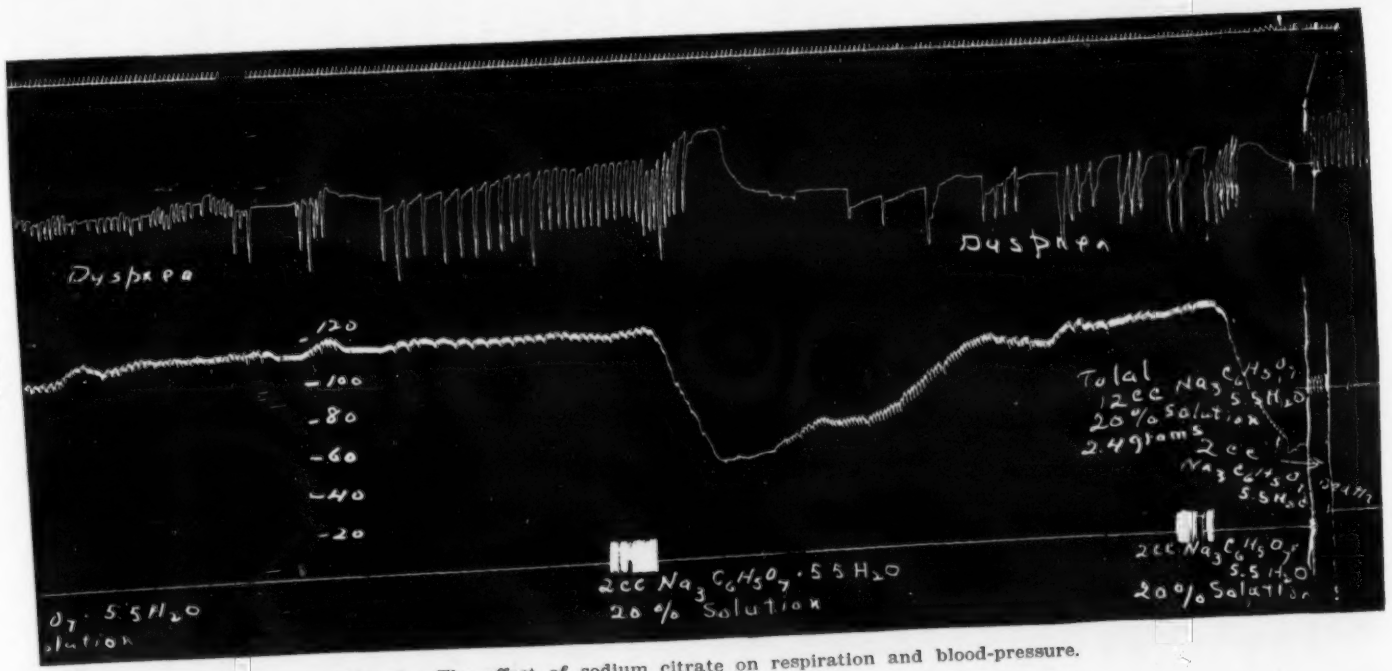


FIG. 1. The effect of sodium citrate on respiration and blood-pressure.

be immediately corrected by the intravenous administration of a 20 per cent solution of calcium chloride. They also produced tetany and prostration in rabbits by injecting 2 cc of an 8 per cent solution of sodium citrate into the auricular vein. The intravenous injection of an equivalent amount of an 8 per cent calcium chloride solution restored the animals to normal within one minute. The authors concluded that the sodium citrate formed a nonionizable calcium citrate which interfered with the mobilization of calcium and produced the condition of tetany, prostration and severe expiratory dyspnea, dilatation of the pupils and, finally, paralysis.

Table II records the results obtained on respiration and blood-pressure as the result of the intravenous injection of a 20 per cent solution of sodium citrate into a male dog weighing 5.49 kg. The initial injection of 0.20 gm caused a fall in blood-pressure from 135 to 103 mm of Hg and a temporary paralysis of the respiratory center. These effects were transient. The second injection of 0.10 gm caused a decrease in blood-pressure from 132 to 122 mm of Hg but an increase in the respiratory rate from 28 to 40 per minute. The third injection of 0.20 gm produced a similar effect; namely, it reduced the blood-pressure from 130 to 112 mm of Hg and increased the respiratory rate from 30 to 50 per minute. The pulse pressure or the difference between the systolic and diastolic pressure was increased from 2 to 4 mm of Hg. Subsequent injections produced in each case a fall in blood-pressure and a temporary holding of the respirations. Tetany and dyspnea appeared after the fourth injection, or after 0.8 gm had been injected. The lethal dose of sodium citrate was 2.0 gm. In general it can be said that this salt has a depressing effect upon blood-pressure and respiration, probably exerting its effect upon the medullary centers. To what extent this is a direct effect of the sodium citrate upon the centers or an indirect effect resulting from the displacement of calcium, it is impossible to state.

Potassium Chloride

Because the presence of potassium ions is essential to the vital functioning of various tissues, the toxicity of potassium chloride is of special interest.

Table III shows the toxic effect of potassium chloride on respiration and blood-pressure as the result of the intravenous injection of a 5 per cent solution into a female dog, weighing 10 kg. Small doses (0.075 gm) caused a rise in blood-pressure. Following the third injection, or a total of 0.250 gm, the blood-

pressure rose to 218 mm of Hg. The lethal dose was 0.850 gm. Following the last injection, which consisted of 0.200 gm, the blood-pressure rose from 180 to 192 mm of Hg and an increase in the respiratory rate from 17 to 49. This was followed in 15 seconds by a rapid fall in blood-pressure to a minimal level of 40 mm of Hg and cessation of respirations with the death of the animal. A postmortem examination of the animal showed the heart to be in a relaxed or dilated state, indicating that it had stopped in diastole (potassium inhibition).

The toxicity of potassium citrate is of interest, because of the similarity it bears to sodium citrate. Potassium citrate is also an anti-coagulant but is not recommended for transfusion purposes.

Table IV gives the results of a 20 per cent solution of potassium citrate injected intravenously into a female dog weighing 8 kg. It is to be noted that in the preliminary record, before the administration of the salt, there was a fall in blood-pressure from 180 to 150 mm of Hg. This fall was due to a venous hemorrhage, which resulted from a perforation of the femoral vein. No significant change in respiration or blood-pressure was apparent as a result of the injection of 0.04 gm of potassium citrate. However, a second injection, constituting 0.10 gm,

TABLE III—*Effect of potassium chloride (5 per cent solution) on respiration and blood-pressure (female dog, 10 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)
8:47	164	14	
8:50	164	14	0.075
8:50:30	182	36	
8:52	170	14	0.075
8:52:30	180	30	
8:53	174	12	0.100
8:53:30	218	48	
8:55	178	12	0.100
8:55:30	188	45	
8:57	180	14	0.150
8:57:15	186	49	
8:58	178	15	0.150
8:58:15	184	56	
9:00	180	17	0.200
9:00:15	192	49	
9:00:30	40	18*	
9:01:15†	40	Ceased	
		Total	0.850

*Respirations very irregular.
†Death.

caused a rise in blood-pressure from 154 to 180 mm of Hg and an increase in the respiratory rate from 170 to 210 per minute. Three minutes following this injection, the blood-pressure had fallen to 146 mm of Hg and the respiratory rate was 200 per minute. This, and subsequent injections, caused an inhibition of the respiratory center and a stimulating effect upon blood-pressure.

A fourth injection, consisting of 0.20 gm of the salt, caused the blood-pressure to rise from 140 to 208 mm of Hg and a slowing of the respiratory rate from 130 to 100 per minute. As a result of the fifth injection, which consisted of 0.40 gm (total of 0.94 gm), the blood-pressure increased from 140 to 180 mm of Hg and the respirations ceased. The animal showed marked tetany, and after cessation of respiration the blood-pressure rose to 226 mm of Hg. The respirations were spontaneously resumed after 30 seconds and gradually increased to the rate of 140 per minute. The final injection of 0.40 gm (total of 1.34 gm) caused a transient rise in blood-pressure from 130 to 142 mm of Hg and a cessation of respiration. The blood-pressure rapidly dropped to 58 mm of Hg and death ensued.

TABLE IV—*Effect of potassium citrate (20 per cent solution) on respiration and blood-pressure (female dog, 8 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)
3:10	180	170	
3:26*	150	170	
3:27	150	170	0.040
3:29:30	154	170	0.100
3:29:45	180	210	
3:30	145	150	
3:32:30	146	200	0.200
3:32:45	225	100	
3:35:30	140	130	0.200
3:36	208	100	
3:37:30	140	130	0.400
3:37:45†	180	Ceased	
3:38	226	50	
3:39	123	140	
3:41	140	140	
3:42:15	130	120	0.400
3:42:30‡	142	Ceased	
3:45§	58		
		Total	1.34

*Hemorrhage from femoral vein.
†Tetany.
‡Tetany.
§Artificial respiration instituted. Animal died, respiration stopping first.

FIG. 2. The effect of potassium citrate on respiration and blood-pressure.

Calcium therapy by intravenous and hypodermic methods of administration has become an important clinical procedure in the treatment of a number of diseases. Calcium chloride is very readily soluble and ionized, which causes its effects to be produced very quickly. On the other hand, calcium chloride is quickly eliminated from the blood. Calcium chloride is one of the most toxic of the calcium salts and will cause sloughing even if only a few drops enter the tissue, hence the necessity of intravenous administration.

Table V records the effects of a 20 per cent solution of calcium chloride injected intravenously into a female dog weighing 9.09 kg. A preliminary injection of 0.20 gm of calcium chloride produced a rise of blood-pressure from 100 to 136 mm of Hg and a slowing of respirations from 32 to 26 per minute. A second injection of 0.20 gm produced a similar effect, namely, a rise in blood-pressure from 118 to 142 mm of Hg and a slowing of respirations from 28 to 21 per minute. Similar effects were noted with each injection. The heart-beats became irregular and were peculiar in having an irregular rhythm, characterized by a very strong beat occurring every five to ten seconds. This strong beat produced a pulse pressure of 50 mm of Hg, which was the difference between the systolic and diastolic pressures. The

TABLE V—*Effect of calcium chloride (20 per cent solution) on respiration and blood-pressure (female dog, 9.09 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	REMARKS
2:31	105	35		
2:36	100	32	0.200	Peculiar heart-beat. Regular pulse pressure
2:36:30	136	26		
2:37	123	26		
2:38	118	21	0.200	Irregular heart-beat disappeared
2:38:30	142	28		
2:38:45	125	21		Periodic, irregular beat reappeared but less frequently
2:41	120	28	0.200	
2:41:30	145	21		
2:42	122	21		
2:43	130	21	0.400	
2:43:30	140	28		Pulse pressure 60 mm.
2:44	18	50		Heart stopped. Respirations continued for about 1 minute after
2:55	18	50		Heart stopped
		Total	1.000	

TABLE VI—*Effect of calcium gluconate (20 per cent in 3 per cent boric acid) on respiration and blood-pressure (male dog, 20 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	PULSE PRESSURE (MM)	REMARKS
3:00	152	16			
3:04	152	21	0.10		
3:04:30	140	35			
3:04:45	156	35			Decrease in amplitude
3:06	146	42	0.10		
3:06:30	150	56			
3:08	150	50	0.20	16	Increased strength of heart-beat
3:11	162	42			
3:13	157	40	0.20		BP. and R. remain fairly constant for next 2 minutes
3:15	160	50	0.40		Rhythmic periodic increase in pulse rate
3:17	162	56	0.40	18	
3:19	170	56	0.60	20	R. weak in amplitude
3:22	172	50		40	
3:23	174	50	0.60	40–50	
3:26	170	70	0.80	10	
3:26:30	185	70		28–30	
3:30	180	63	1.00	18	R. shallow
3:30:30	197	63		30–35	
3:32	180	56	1.00	20	R. shallow and weaker
3:34	184	36	2.00	18–20	
3:35	210	28–54			
3:36	190	70	3.00		
3:37	224	63		20–46	
3:37:30	216	21		64	R. very marked increase in amplitude
3:38	210		4.00	70	
3:40	217	49			
3:43	225	35	4.00	18	
3:45	210	25	5.00	16	
3:47	220	50	6.00	16–18	
3:50	220	28	10.00	18	
3:52	182	30			R. marked increase in amplitude
3:54	203	28	10.00	16	
3:55	183	30			R. marked increase in amplitude
4:00	210	26	4.00	14–16	
4:25					Following the injection of 53.4 gm the animal was doing well
		Total	53.4		

BP. = blood pressure.
R. = respirations.

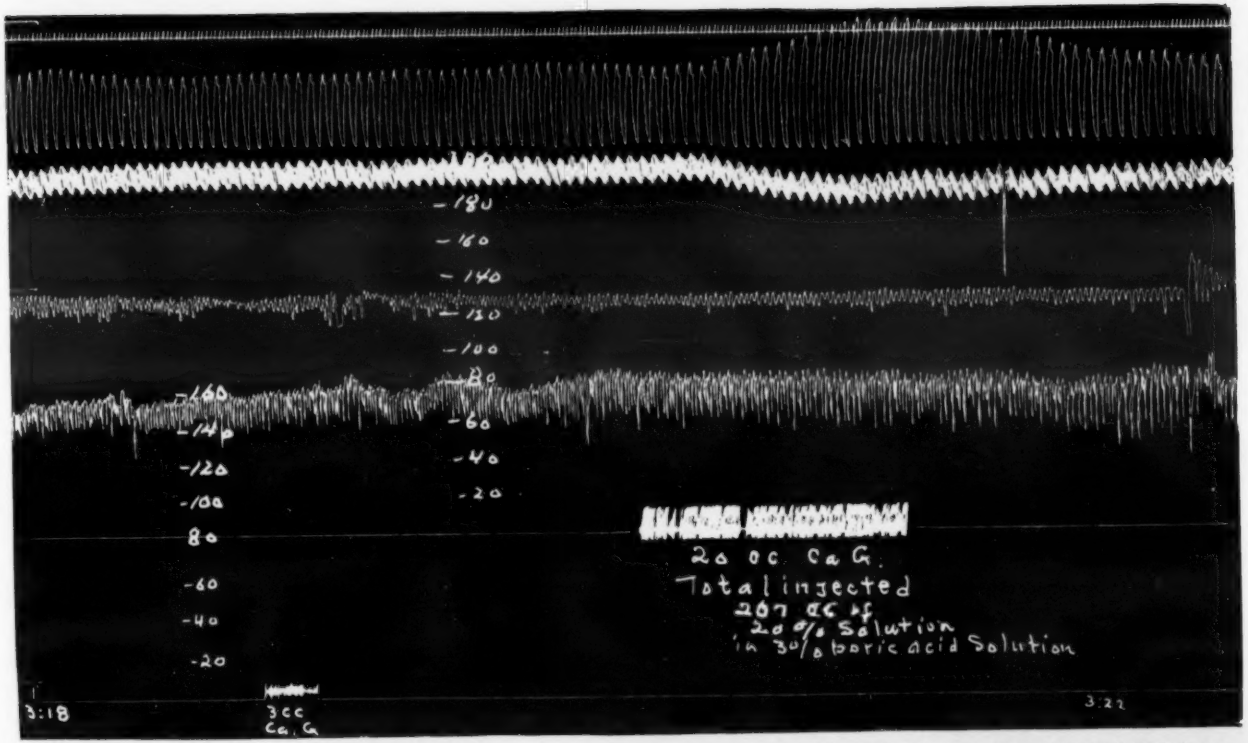


FIG. 3. The effect of 20 per cent calcium gluconate. This figure shows the effect of 0.20 gm of calcium gluconate on respiration and blood-pressure measured from the bottom base-line. It also shows the effect of a total of 53.4 gm of calcium gluconate on respiration and blood-pressure measured from the second base-line, which is the second line from the bottom.

lethal dose was 1.0 gm injected over a period of seven minutes. The last injection (0.4 gm) caused a temporary rise in blood-pressure to 140 mm of Hg followed by a sudden drop to 18 mm. The rate of respirations increased from 21 to 28 and later to 50 per minute. This increase is probably due to a reflex stimulation of the respiratory center, due to the failure of the circulatory system, producing asphyxia of the tissues. Autopsy of the animal revealed that the heart had stopped in a contracted state (calcium rigor).

Table VI records the data obtained on respiration and blood-pressure as the result of the intravenous injection of a 20 per cent solution of calcium gluconate dissolved in 3 per cent boric acid solution into a male dog weighing 20 kg. The total injection of 53.4 gm over a period of one hour in doses varying from 0.10 to 10.0 gm failed to cause the death of the animal. The blood-pressure was 152 mm of Hg at the beginning of the experiment and 210 at the end of one hour and 25 minutes.

Table VII records the results obtained on respiration and blood-pressure as the result of the intravenous injection of a 30 per cent solution of calcium gluconate into a male dog weighing 13.63 kg. In this case, the total injection of 15 gm, in doses varying between 1.5 and 4.5 gm over a period of 18 minutes, caused the heart and respirations to cease, the heart stopping one minute before the respirations.

TABLE VII—*Effect of calcium gluconate (30 per cent solution in 4 per cent boric acid solution) on respiration and blood-pressure (male dog, 13.63 kg).*

TIME	BLOOD PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	PULSE PRESSURE (MM)	REMARKS
2:48	156	21		6	
2:53	145	28	1.50	8	
2:54	154	35	1.50	10	
2:56	154	40	3.00	8	
2:57	150	45	4.50	8	
2:58	100	63		4–6	R. shallow
3:00	162	54		2	R. increased in amplitude
3:02	172	28	1.50	2	
3:05	180	38	3.00	2	
3:06	42	56			Heart stops. R. markedly increased in amplitude
3:07					R. cease
		Toral	15.00		

R. = respirations.

Fig. 20.—Effect of 0.20 per cent calcium gluconate. This figure shows the effect of 0.20 gm of calcium gluconate on respiration and blood-pressure measured from the bottom base-line. It also shows the effect of a total of 53.4 gm of calcium gluconate on respiration and blood-pressure measured from the second base-line, which is the second line from the bottom.

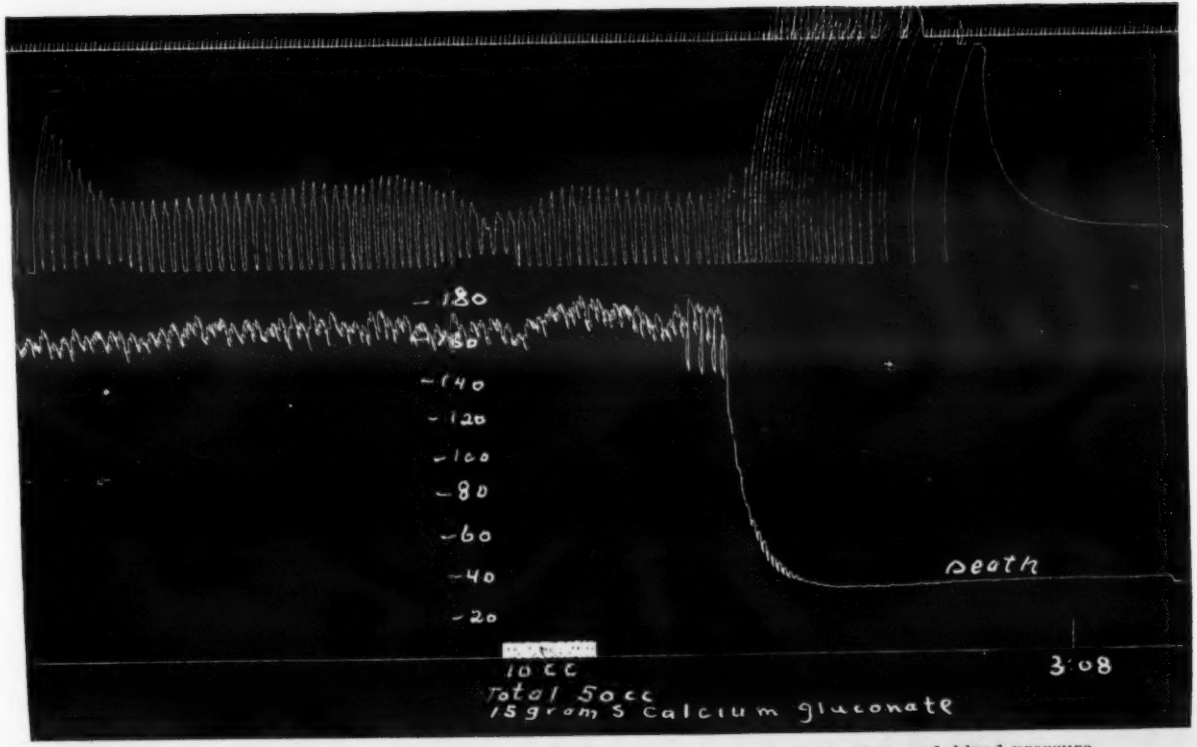


FIG. 4. The effect of 30 per cent calcium gluconate solution on respiration and blood-pressure.

Figures 1 to 4 show typical sections of tracings recording the effects of sodium citrate, potassium citrate, and calcium gluconate on respiration and blood-pressure.

SUMMARY

1. Sixty and six-tenths gm of sodium chloride in 30 per cent solution injected intravenously over a period of 17 minutes proved fatal to a dog weighing 18.18 kg. This is equivalent to 3.30 gm per kg body weight. Autopsy revealed marked edema of the lungs and liver. The observations from this experiment indicate that the toxicity of sodium chloride is effected in three ways: (1) The osmotic pressure effect, which increased blood-volume and, hence, blood-pressure; (2) a direct effect upon the medullary centers at first stimulating and later paralyzing these centers, and (3) increasing the permeability of the cell membranes, causing fluid to escape more readily into the tissue spaces.

2. Sodium citrate has a depressing effect upon blood-pressure and respiration, exerting its effect upon the medullary centers either by direct effect of the sodium citrate or by affecting the calcium. Two grams in 20 per cent solution injected over a period of 18 minutes caused the death of a dog weighing 5.49 kg. This is equivalent to 0.364 gm per kg of body weight.

3. Eighty-five hundredths gm of potassium chloride in 5 per cent solution proved sufficient to cause the death of a dog weighing 10 kg (0.085 gm per kg.) In general each administration of KCl was followed by a rise in blood-pressure and a slowing of respiration, probably through a direct action upon the medullary centers.

4. Potassium citrate in toxic doses has an inhibitory effect upon respiration and a stimulating effect upon blood-pressure. One and thirty-four hundredths gm was the lethal dose for a dog weighing 8 kg or 0.167 gm per kg of body weight.

5. One gram of calcium chloride proved to be a lethal dose for a dog weighing 9.09 kg or 0.11 gm per kg. The injections produced an irregular heart-beat with the heart stopping in systole.

6. Fifty-three and four-tenths gm of calcium gluconate in 20 per cent solution injected over a period of one hour failed to produce toxic symptoms in a dog weighing 20 kg; whereas, when a 30 per cent concentration was employed, 15 gm proved to be a lethal dose for a dog weighing 13.63 kg. In other words, when a 20 per cent solution was used, 2.67 gm per kg failed to produce

Fig. 4. The effect of 30 per cent calcium gluconate solution on respiration and blood-pressure.

toxic symptoms; but when a 30 per cent solution was used, 1.10 gm per kg proved fatal.

REFERENCE

[1]Petersen, W. E., Hewitt, E. A., Boyd, W. L., and Brown, W. R.: Artificially induced hypoglycemia and hypocalcemia in the cow and the relationship to parturient paresis or milk fever. Jour., A. V. M. A., lxxix (1931), n. s. 32 (2), pp. 88-93.

Bureau Transfers

DR. HILBORN H. GROAT (K. S. C. '21), from Topeka, Kan., to Denver, Colo., on meat inspection.

DR. JOHN H. COLLINS (O. S. U. '35), from New York, N. Y., to South Omaha, Neb., on meat inspection.

DR. RAYMOND V. SCHOENTRUP (O. S. U. '36), from Rockymount, Va., to Chicago, Ill., on meat inspection.

DR. RAY HOEFLING (Cin. '11), from South Saint Paul, Minn., to Cincinnati, Ohio, on meat inspection.

DR. THOMAS J. MUXLOW (K. S. C. '30), from Watertown, S. Dak., to South Saint Paul, Minn., on meat inspection.

DR. F. A. BARBER (K. C. V. C. '12), from The Dalles, Ore., to Boise, Idaho, on hog cholera and special disease control.

DR. C. H. FAUKS (McK. '07), from Oklahoma City, Okla., to Pierre, S. Dak., in charge of tuberculosis eradication.

DR. VIRGIL A. YOUNT (Colo. '36), from Oklahoma City, Okla., to Kansas City, Kan., on meat inspection.

DR. NORMAN W. ACKERMAN (Iowa '15), from Chicago, Ill., to Buffalo, N. Y., on meat inspection.

DR. DALYS L. BERRY (K. S. C. '32), from Chicago, Ill. to Fort Worth, Texas, on meat inspection.

DR. WALLACE W. BOGGIE (Iowa '21), from Mitchell, S. Dak., to Fort Dodge, Iowa, in charge of meat inspection.

DR. JOHN L. CORE (Gr. Rap. '15), from Albany, N. Y., to Wheeling, W. Va., on meat inspection.

DR. GEO. E. GILBERT, JR. (T. H. '16), from South Saint Paul, Minn., to Eau Claire, Wis., on meat inspection.

DR. ALBRO A. LEMERY (K. C. V. C. '08), from Newark, N. J., to Kansas City, Kan., on meat inspection.

DR. FRANK J. LINGO (Cin. '08), from Albuquerque, N. Mex., to San Antonio, Texas, on yards inspection and Bang's disease control.

DR. IRVINE LEWIS, JR. (Colo. '20), from Lewiston, Idaho, to Spokane, Wash., on meat inspection.

DR. CHARLES A. HAZZARD (K. S. C. '12), from Jersey City, N. J., to Newark, N. J., on meat inspection.

DR. JOSEPH M. JEHLE (Chi. '11), from Lafayette, Ind., to Indianapolis, Ind., in charge of meat inspection.

DR. ARTHUR L. STIGERS (Iowa '30), from Sioux City, Iowa, to Des Moines, Iowa, on meat inspection.

DR. A. K. KUTTLER (San Fran. '17), from Fort Worth, Texas, to Boise, Idaho, in charge of tuberculosis eradication.

X-RAY PROTECTION*

By G. E. WANTZ, *General Electric X-Ray Corporation, Chicago, Ill.,*
and E. J. FRICK, *Kansas State College, Manhattan, Kan.*

The veterinary profession of recent years has become x-ray conscious and rightfully so, as there is an important, necessary, useful field for its use in veterinary medicine. However, the use of the x-ray is not entirely without its hazards.

While attempting to demonstrate at an x-ray clinic at the Oklahoma City meeting of the American Veterinary Medical Association in 1935, the authors were impressed with the fact that at least two visiting veterinarians were definitely showing finger signs of early x-ray burns and were entirely ignorant of what was causing the trouble.

The purpose of this article is not to cause undue apprehension because, if x-ray apparatus is used properly and the well-known means of protection (which are simple and easily employed) are utilized, there is *no reason or excuse* for either operator or patient receiving either a temporary or permanent injury or burn. The advantages of the use of the x-ray are all too obvious. That some disadvantages also need to be considered seems apparent.

An outline of the important hazards to be understood in the application of x-ray to veterinary practice may serve to prevent some of the detrimental experience which our colleagues in the medical field have had.

There are two "hazards" commonly associated with the operation of x-ray equipment, one being electrical shock, and the other the damage done as the result of the cumulative effects of x-ray on the body tissues. The first danger may occur due to the necessity for high voltage at the x-ray tube for the production of x-radiation. This hazard is a real one, but is found only in those equipments of the so-called "open wire" type, in which the x-ray tube is energized from conductors not designed to give high voltage protection.

This fact should be considered when one is tempted to purchase the many second-hand, non-shock-proof, non-portable equipment, on the market. Such apparatus is not safe from an electrical standpoint, either to the operator or the patient, and is not satisfactory for veterinary practice.

*Received for publication, March 6, 1937.

Its seriousness must be realized by all operators of such apparatus, for the result of contact with, or shock from, such high voltage is not only extremely unpleasant but, in some instances, is very injurious or may be fatal.

Modern equipment of the capacities generally suitable for veterinary work is of a construction which entirely eliminates this form of hazard. The "open wire" type, mobile machine has been superseded by a shock-proof unit in which the x-ray tube, together with the high voltage transformer, is immersed in oil in a metallic container which acts as a perfect shield, entirely eliminating any possibility of shock.

Large equipment of the permanent installation type may be made relatively safe by the use of a shock-proof x-ray tube unit which completely protects the operator against electrical hazard from the tube or connecting cables to the point where they join the overhead system, beyond which, because they are several feet above the head of the operator, there should be no material danger.

The other hazard—a very important one, especially to the small-animal practitioner—arises from the absorption of x-radiation by the operator, patient, or anyone else in its field of influence and, because its effects are not apparent immediately after exposure takes place, it becomes necessary to have a complete understanding of what it is and the precautions necessary for adequate protection.

Unfortunately, since this hazard is from the effects of radiation absorption (and which radiation must be emitted from the tube for practical usage in x-ray examination), it follows that complete protection against its effects cannot be designed into the equipment by its manufacturer. *The presence of x-radiation is, of course, neither seen, felt, nor heard—and since its insidious reaction is not recognized for days, weeks, or months, to fail to take proper precautions* is merely inviting trouble.

The Nature of X-Rays

X-radiation originates at the focal spot of the x-ray tube, may be radiated in all directions from it, and is sometimes described as "invisible light." The typical tube for veterinary or medical usage has the face of the anode set at an angle 20° from the perpendicular. From this, radiation is emitted in a completely hemispherical direction. As a baffle prevents visible light from shining backward, so, too, does the form of the anode prevent x-rays from traveling in this direction.

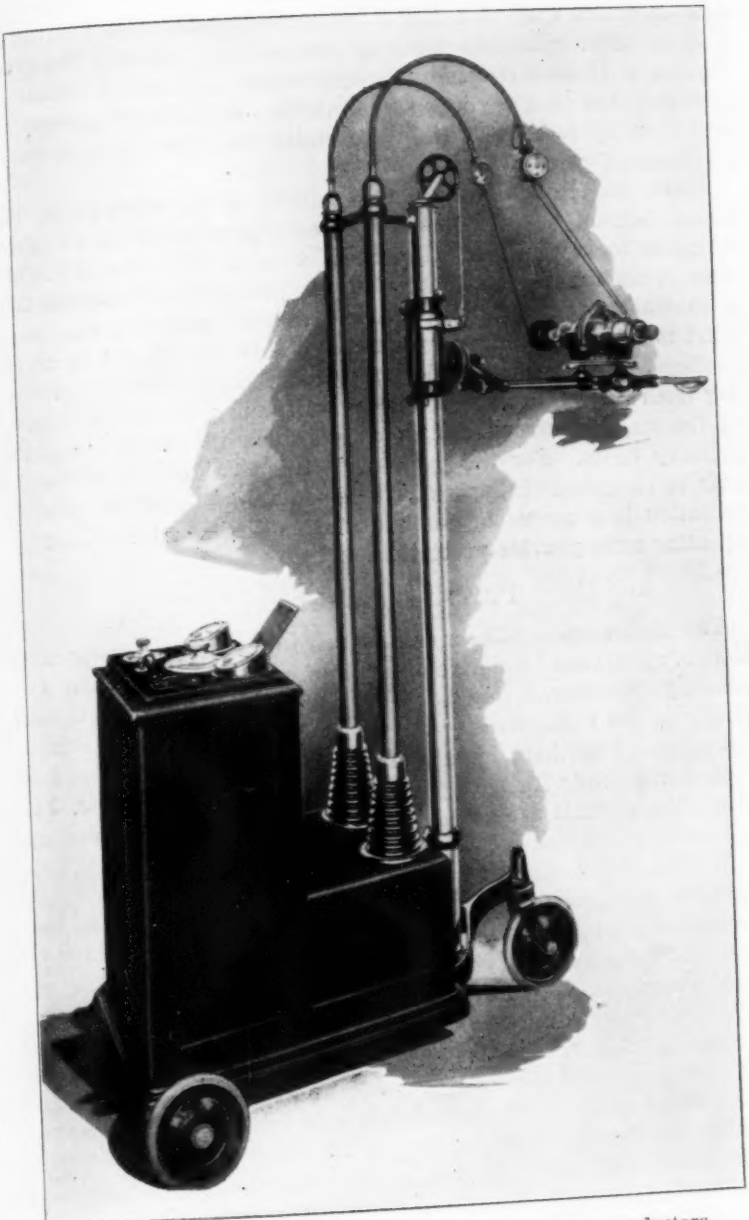


FIG. 1. Mobile x-ray unit with exposed high-voltage conductors.

Modern x-ray tube and equipment design further limits the area in which such radiation is present by placing a barrier of lead or other relatively x-ray-opaque material around the tube. Having a limited opening through which the useful radiation emerges, modern x-ray apparatus provides a means of generating and emitting radiation within the limits described by a comparatively small cone.

Since reactions to radiation result from its absorption, the above fact might suggest that the only precaution to be taken would be to stay outside this cone of x-rays. This would be true were it not for the fact that, wherever primary radiation strikes a substance, that substance becomes a new emitting source of what is called "secondary radiation." This travels out in *all* directions. The quantity and quality of secondary radiation arising from these sources depends upon the physical characteristics of the material struck, and of course upon the intensity of the primary beam. For the purpose of this article it is necessary only to recognize that any substance in the path of the primary radiation is a source of secondary radiation against which the operator must provide protection.

PHYSIOLOGICAL EFFECTS

The physiological effects of x-ray are dependent, among other things, upon, the cumulative absorption over a given period. Fortunately, however, living body is capable of throwing off these effects if the total quantity absorbed within a given time is not too great. Elsewhere in this article are given the generally accepted safe limits for both patient and operator. (In animal patients these limits are as yet only approximated.) These should not be exceeded unless the particular circumstances warrant.

Whether by cumulative absorption or from continuous exposure, an overdose of x-ray produces blood changes, loss of hair, malignancies, malfunction of internal organs, erythemas, and the like, the latter usually referred to as "x-ray burns." The first manifestation of too much exposure to a given part may be a loss of hair, or reddening of the skin. However, all of these do not necessarily follow, for effects are tied up completely with the rate of absorption and the character of the absorbed radiation. As with those upon deep-seated organs, skin effects vary from those which time and freedom from radiation will correct to those which are so gross as to require surgical or other intervention, and those leading even to permanent injury or worse.

As concerns the operator, the hands are most likely to suffer, as it is altogether too common a practice for him to place his un-

protected hands in the x-ray field for the purposes of immobilization or palpation. The amount of radiation absorbed in a single examination would rarely be dangerous, but since the effects are associated with cumulative absorption, *every precaution to avoid unnecessary exposure should be taken.*

With present-day knowledge and the availability of protective materials, the consequences of too much radiation can be associated only with gross carelessness or ignorance on the part of the operator, unless those results are definitely sought, as may be the case in the x-ray treatment of certain conditions.

It may be truthfully said that the operator should be more concerned with his own protection than with that of his patient, al-

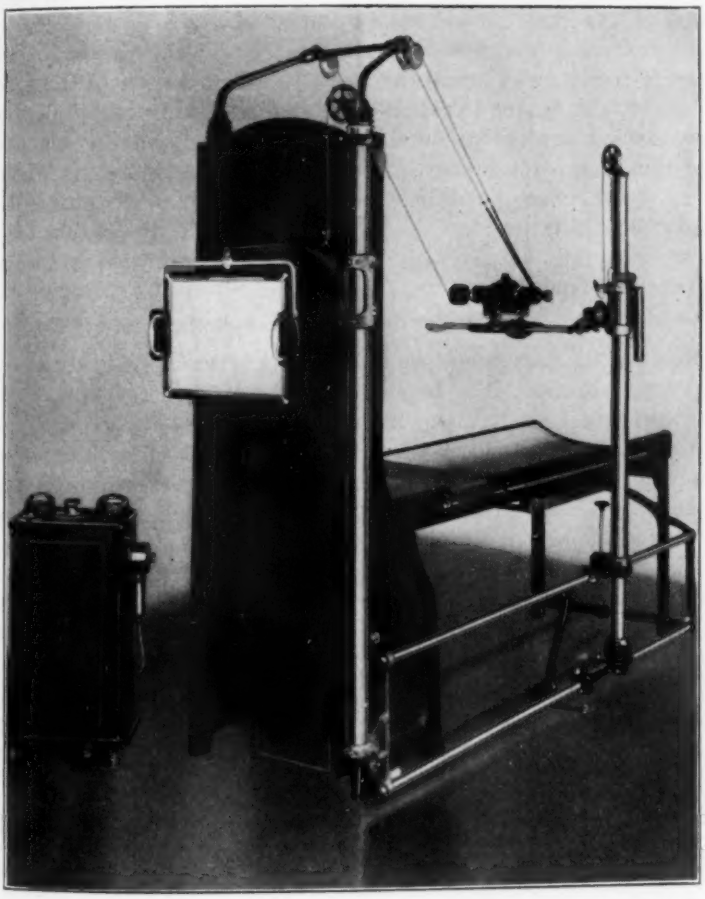


FIG. 2. A permanently installed x-ray unit with exposed high-voltage conductors.

though this is not to be interpreted as advocating a deviation from the altruism for which the profession is noted.

The average x-ray examination—roentgenographic, fluoroscopic, or both—can be conducted with a radiation absorption well within the safe tolerance of the patient. Moreover, the average patient presents himself for x-ray examination but a comparatively few times, if for more than once. Thus such a patient will receive a quantity of radiation the effects of which are well within the body tolerance.

The operator, however, particularly if doing an appreciable amount of fluoroscopy and unless proper protective measures are taken, is constantly exposed to small amounts of radiation. Thus it is *more important* to consider operator- than patient-protection, although the latter should not be neglected.

The extent to which one should go in providing protection for himself will depend upon the quantity of work to be handled. However, a safe rule to follow is that of utilizing ample precautions during fluoroscopy by the use of suitable protective devices and minimum examination times. For radiography, as for all x-ray work, where possible, one should remain well outside the active field of radiation. As with visible light, the intensity of x-radiation falls off inversely with the square of the distance as one recedes from its source.

Generally Accepted Safe-Exposure Limits

Because the patient is exposed more particularly to direct, or primary, radiation, it is impossible and practical to express safe operating limits in terms of milliampere-seconds for given filters, voltages and distances. With most equipment a table is provided, applying to that particular unit, which gives necessary instructions for its use. If such a chart is not available, the manufacturer should then be asked for his advice.

The radiation tolerance of the operator, which, it must again be said, is of particular importance, cannot be easily expressed in readily measured units, although in later paragraphs certain recommendations will be found.

It is probably safe to say, however, that for roentgenography it is suitable to remain well outside the active field and away from the patient, while for fluoroscopy certain following general suggestions should be adhered to.

Recommendations of the International X-Ray and Radium Protective Commissioners

Following are pertinent excerpts from the recommendations for x-ray protection as revised by the International X-Ray and Ra-

dium Protective Commission at the Fourth International Congress of Radiology, Zurich, July, 1934, and extracted from *Radiology*, Vol. 23, No. 6, December, 1934.

1. The dangers of over-exposure to x-rays and radium can be avoided by the provision of adequate protection and suitable working conditions. It is the duty of those in charge of x-ray and radium departments to insure such conditions for their personnel. The known effects to be guarded against are:

(a) Injuries to the superficial tissues.

(b) Derangements of internal organs and changes in the blood.

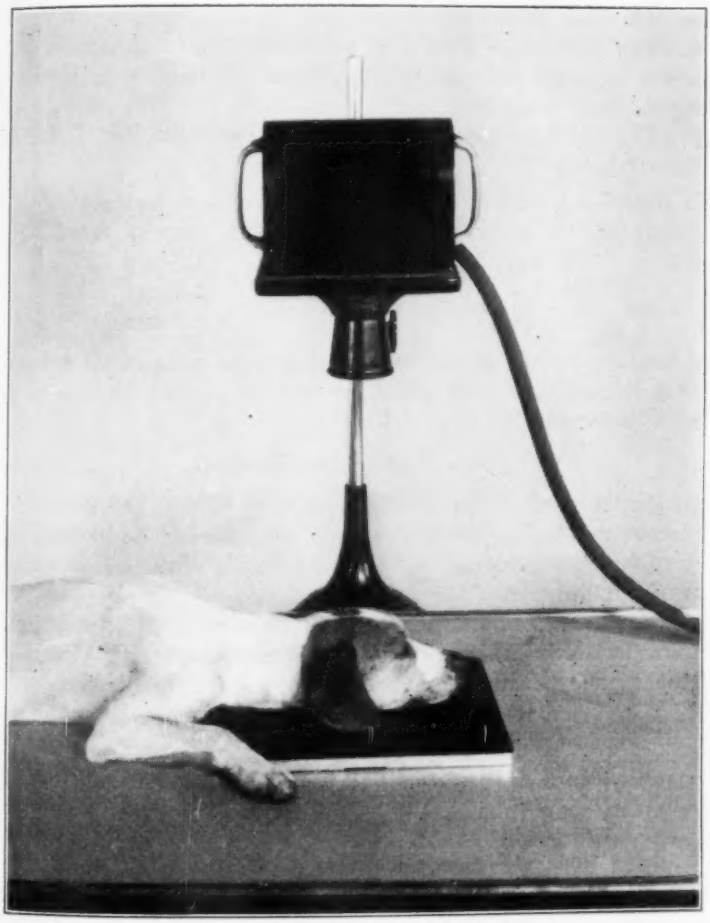


FIG. 3. A portable x-ray unit which is shock-proof and electrically safe because all high-voltage parts are immersed in oil within a single metal container.

The evidence available at present appears to suggest that under satisfactory working conditions a person in normal health can tolerate exposure to x-rays to an extent of about 0.2 international roentgens (r) per day. On the basis of continuous irradiation during a working day of seven hours, this figure corresponds to a dosage rate of 10^{-5} r per second. The protective values given in these recommendations are generally in harmony with this figure under average conditions. No similar tolerance dose is at present available in the case of radium gamma rays.

2. An x-ray operator should on no account expose himself to a direct beam of x-rays.

3. An operator should place himself as remote as is practicable from the x-ray tube.

4. The x-ray tube should be self-protected or otherwise surrounded as completely as possible with protective material of adequate lead equivalent.

5. The following lead equivalents are recommended under average conditions:

X-rays Generated by Peak Voltages Not Exceeding	*Minimum Equivalent Thickness of Lead*
75 kv.	1.0 mm
100 kv.	1.5 mm
125 kv.	2.0 mm

The lead equivalent of a given thickness of protective material is that thickness of lead which is equally opaque to x-rays, excited at some specific peak voltage.

A. Diagnostic Work

6. In the case of diagnostic work with other than completely protected tubes, the operator should be afforded additional protection from stray radiation by a screen of a similar lead equivalent of 1 millimeter.

7. Screening examinations should be conducted as rapidly as possible with minimum intensities and apertures. Palpation with the hand should be reduced to the minimum.

8. The lead glass of fluorescent screens should have the protective values recommended in paragraph 5.

9. In the case of screening stands the fluorescent screen should, if necessary, be provided with a protective "surround" so that adequate protection against direct radiation is afforded for all positions of the screen and diaphragm.

10. Screening stands and couches should provide adequate arrangements for protecting the operator against scattered radiation from the patient.

11. Protective gloves, which should be suitably lined with fabric or other material, should have a protective value not less than 1/3 mm lead throughout both back and front (including fingers and wrist). Protective aprons should have a minimum lead value of 0.5 mm.

B. Treatment

12. In the case of x-ray treatment the operator is best stationed completely outside of the x-ray room behind a protective wall of a minimum lead equivalent of 2 mm. This figure

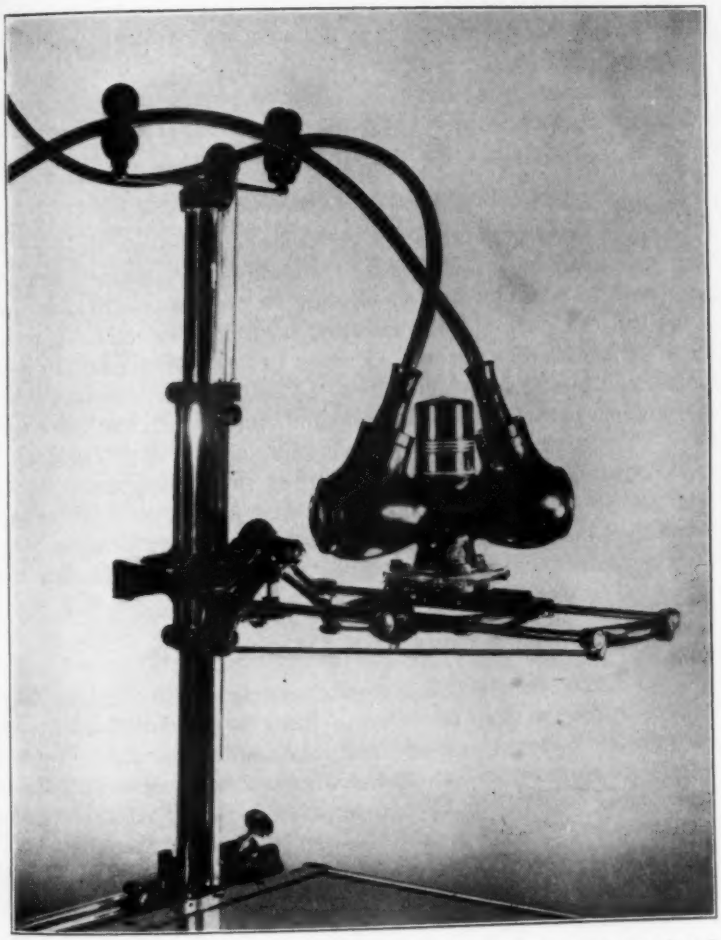


Fig. 4. A permanently installed x-ray unit which is shock-proof and electrically safe because tube is immersed in oil in a grounded metal container and the high-voltage cables are shock-proof.

should be correspondingly increased if the protective value of the x-ray tube enclosure falls short of the values given in paragraph 5. In such event the remaining walls, floor and ceiling may also be required to provide supplementary protection for adjacent occupants to an extent depending on the circumstances.

13. Inspection windows in screens and walls should have protective lead values equivalent to that of the surrounding screen or wall.

14. In those cases in which an ex-ray tube is continuously excited and treatment periods are regulated by means of a shutter, some form of remote control should be provided for the shutter, to insure that the operator is not exposed to direct radiation while manipulating the shutter or filter.

Efficient safeguards should be adopted to avoid the omission of a metal filter in x-ray treatment. To this end some means of continuously measuring the emergent radiation is recommended.

PROTECTIVE ACCESSORIES

Manufacturers generally design their equipment to give as much protection as possible, but it should be recognized that no amount of built-in protection eliminates the necessity for precaution on the part of the operator, both in protecting himself and in protecting the patient. Proper protection is easy to have and use without discomfort, either to self or patient, and without handicapping the satisfactory and successful use of equipment. For this purpose there is a complete line of protective devices, all relatively cheap, although they must be good.

In addition, sound advice *may be had* and *should be sought* from those manufacturers or suppliers who have sufficient experience in the field to permit their giving such assistance.

More Articles by Doctor Foster

Dr. J. P. Foster, of Minneapolis, continues to contribute to *The Thoroughbred Record*. One of his recent contributions was a discussion on equine conformation, a subject which he also presented before members of the Minneapolis Saddle and Bridle Club. Other recently published articles included discussions on "Curb," "Some Thoughts on Breeding," "Problems on Selection," "Navicular Disease," and "Equine Breeds and Heredity." He also wrote "Random Observations," "Life's Span," and "Commentaries," in which he discussed resistance to disease and preventable accidents, and "Nelson, the Northern King," the story of a famous trotting stallion of the nineties.

THE PROPHYLACTIC VACCINATION OF DOGS AGAINST RABIES IN A PUBLIC HEALTH PROGRAM*

By C. E. De Camp, *Scarsdale, N. Y.*

Rabies is one of the most obstinate and dangerous diseases existing throughout the world. It affects both human and animal populations, and one infected animal may be a menace to a large community. Because of this disease being a local, state, national and international problem, its control for the welfare of public health is important. Although rabies may exist in all animals, it is most prevalent in and disseminated mostly by dogs, and control measures must center around this animal.

The control of rabies in the dog consists of the muzzling and leashing of all animals while not on the owners' property, the collection and destruction of all stray, unlicensed, unmuzzled dogs, and the vaccination of *all* dogs against this disease. This paper will review the experiences and opinions of representative health workers on the value of the single, antirabic vaccination of dogs, once yearly, as *part* of a program for controlling the disease.

Pioneer Work

One of the first reports of favorable results obtained by using a single injection of attenuated virus for the protection of dogs against rabies was made in 1921, by two Japanese investigators, Umeno and Doi.[1] Experimental studies by Schoening, of the U. S. Department of Agriculture, reported in 1925,[2] indicate that there are more than one strain of street rabies virus in the United States. This work was studied in 1928 and reported upon by Broerman and Edgington,[3] with the following conclusions: Street virus of rabies from different sources varies in virulence. The single-injection method of vaccinating dogs against rabies is commendable from a practical standpoint. However, vaccination cannot be depended upon to eradicate rabies until a method is devised that will protect dogs against all strains of street rabies virus.

A more recent report by Schoening[4] points out that in controlling outbreaks of rabies it is essential that the cycle, by which the disease is propagated from one animal to another, be broken so that the disease ceases when the last infected animal dies.

*Received for publication, July 26, 1937.

The successful use of a prophylactic vaccine as a *sole* means of controlling rabies must be predicated on the assumption that (1) the vaccine is 100 per cent effective, and (2) that every dog is treated with such vaccine. As far as is known, no biological product has yet been developed that is 100 per cent effective, rabies vaccine being no exception, and the ever-present stray dog precludes the possibility of treating the entire canine population.

Vaccine Extensively Used in the United States

Following the work of the Japanese investigators, Umeno and Doi,[1] the prophylactic, single-injection method of vaccinating dogs was introduced into the United States as an added weapon in the control of rabies. Its extensive use is indicated by the records of the Division of Virus-Serum Control of the U. S. Bureau Animal Industry, which has jurisdiction over biological houses operating under federal veterinary licenses. From January, 1933, to January, 1934, approximately 3,700,000 doses of this type of vaccine were prepared by such commercial houses.

The use of prophylactic vaccination in conjunction with other measures in controlling rabies is predicated on the reduction of the susceptibility of dogs to the disease. The extent to which vaccination will be an aid depends on the efficacy of the vaccine and its proper and intelligent administration. Since this is a technical procedure, it should be in the hands of the veterinarian. To be of value, a vaccine need not be 100 per cent effective, but it should be reasonably effective, it should be a safe procedure, and the limitations of the method should be recognized.

In a report by Schoening,[5] of the U. S. Bureau Animal Industry, it was stated that while the fixed virus entering into the vaccine is attenuated, it is capable, under certain circumstances, of inducing the fixed-virus disease, a feature quite objectionable in a prophylactic vaccine. Biological houses were required to change their methods of production so that the virus entering into the vaccine is killed by phenol or chloroform. Federal regulations require that the vaccine be injected subdurally into rabbits (most rigid test available) as a test for the viability of the virus before the vaccine is marketed. If prepared properly, it should contain no material capable of producing disease. This is the type of vaccine that is now, and for some years has been, in use commercially in this country. The Bureau's experimental work on this type of vaccine was reported by Schoening.[6, 7] These experiments, while limited, indicated that resistance to rabies could be increased in many dogs by a single injection of

vaccine. This degree of resistance and its duration vary. Appreciable immunity in some dogs may persist for at least a year. No longer immunity should be expected.

Unfavorable Reactions Not Frequent

Reports from the field use of rabies vaccine in dogs indicate that many hundreds of thousands of dogs have been vaccinated with no untoward effect from the vaccine itself. Occasional reports indicate that unfavorable reactions occur following the injection, which for the most part have been transitory. No generally satisfactory explanation as to the cause of these adverse reactions is available, except that the affected animal is susceptible to that type of foreign protein.

While field evidence on the use of rabies vaccine indicates that unfavorable reactions occur infrequently, the fact that they can, and do, occur should be recognized. Much of the opposition by dog-owners to vaccination is based on a fear of these reactions. Dog-owners are entitled to a frank appraisal of the vaccine. There is no doubt that the vaccine has been unjustly accused of causing trouble in ways in which it was in no way responsible, as obscure conditions may occasionally exist at the time of vaccination.

The basis upon which the value of a biological should be determined is, first, the results which can be demonstrated by animal experimentation, and, secondly, the information gained from carefully evaluated clinical applications. The following reports from clinical sources are worthy of careful consideration.

Results in Japan

The prophylactic vaccination of dogs against rabies has proved a most practical and effective measure for controlling the disease in Japan.[8] The method was introduced in 1918 and has been made compulsory in nearly all parts of the country. Reports from 1923 to 1925 are not complete because the earthquake made control measures ineffective. The number of cases of rabies has declined from 1,041 in 1918, to 60, in 1930. The latter occurred chiefly in unvaccinated dogs. Of 1,699,518 dogs vaccinated, only 301 dogs (0.018 per cent) contracted the disease. The vaccine used corresponds to that produced commercially in the United States.

Surgeon General Orders Dogs Vaccinated

In 1928, after studying the rabies problem, the Surgeon General of the U. S. Army ordered that all dogs on military reserva-

TABLE I—*Antirabies vaccination of animals in U. S. Army posts (Surgeon General's Office).*

YEAR	DOGS VACCINATED	CASES OF RABIES	
		VACCINATED	NOT VACCINATED
1929	1,420	2	0
1930	4,012	3	4
1931*	4,673	0	5†
1932	4,474	9	10
1933	4,255	1?	9‡
1934	4,482	0	12
1935	4,586	4	20
Totals	27,902	19	60

*15 cases in San Antonio.
†Includes 1 cow and 1 horse.
‡Includes 1 cow.

tions of the United States should be treated annually with rabies vaccine. This procedure has continued down to the present time. The figures in table I are of interest.

RESULTS IN DETROIT

Many communities have ordinances requiring the annual vaccination of dogs before a license is issued. Detroit, Michigan, is an example. The results of following this practice are conclusively described in a bulletin from the Health Department of that city, dated May, 1929, and entitled: "Rabies, Consider the Dog." This report carried a summary for four years, 1925-28. The purpose of the study was to collect the recent history of rabies in Detroit, with particular reference to the year 1928, and to determine, if possible, the relative value of vaccination of dogs as a protection against this disease. The following statements in this bulletin are significant:

	In 1928	*Cases of Rabies*	*Cases per 1000*
Vaccinated	28,000	10	0.36
Unvaccinated	98,239*	441	448.91
Totals	126,239	451	449.27

In a comparison of the death-rates in the vaccinated and unvaccinated dogs, the following figures are shown:

	Death-rate (rabies) per 100,000 dogs
Unselected	358
Unvaccinated	449
Vaccinated	35.7

*Estimated.

From these figures, it may be concluded that an unvaccinated animal is 12.5 times more likely to contract rabies than a vaccinated animal.

The combined death-rate in 1928 from six of the common communicable *human* diseases was 134.8. This statement is made in the report:

> To those interested in the conservation of dog life alone, these figures should have a dramatic appeal. The comparison with the rates for *human* diseases shows in a startling manner the seriousness of the situation from the canine point of view. The death rate from rabies among dogs is nearly three times that for all the common communicable diseases of humans put together. The lesson to be learned from a comparison of the vaccinated with the unvaccinated is obvious.

Of the ten cases of rabies which occurred in vaccinated dogs in 1928, it was possible to obtain the exact date of vaccination in only three cases. From this information it was concluded that sufficient time had not intervened following vaccination for protection to be established.

Reports from an unselected group of other cities express the belief that the annual vaccination of dogs against rabies is an important part of the rabies control program.[9-13]

Opinions of Veterinary Colleges

An inquiry on this subject was directed to each of the twelve veterinary colleges in North America. Replies received from nine show: One school has used several thousand doses in the clinic, and the community (in a southern state) has recently gone through a widespread outbreak of virulent rabies. Not a single dog given the preventive treatment developed the disease, while 100 unvaccinated animals died of rabies.

Seven schools had not had an opportunity to study the prophylactic value of rabies vaccine to any great extent. Of this number three schools favored its use, based on reports of its application in neighboring cities and in the U. S. Army. One school, not in the group of six mentioned, stated the vaccine was not 100 per cent effective, but the school was not opposed to its use. One school had had no experience with it, so expressed no opinion. Three schools did not have sufficient experience with it or information to express an opinion.

One school referred to a report made in veterinary literature of an experimental study of rabies vaccine under laboratory conditions. The findings lead to the conclusion that rabies cannot be controlled following this procedure (compulsory vaccination). The findings outlined in this report have not been substantiated

by the studies of other laboratory investigators by the use of antirabies vaccine under field conditions. Quite the contrary has been shown, namely, that rabies vaccine in field use, particularly where a virulent virus is present in a rabies outbreak, has given satisfactory results when the vaccination of dogs was a part of the public health program to control the disease.

CONNECTICUT AND MASSACHUSETTS

In our neighboring states of Connecticut and Massachusetts, the annual vaccination of dogs against rabies has been practiced for more than ten years. Not only is the continued employment of this procedure evidence of the favorable results obtained, but figures from the health departments of these states bear out this fact. In Connecticut, antirabies vaccination was recommended by the Commissioner on Domestic Animals in 1922 because of the fact that rabies was very prevalent in that state. In the biennial report of the Commissioner, in 1924, it is stated:

> Antirabies vaccination is beyond the experimental stage and is no doubt one of the very best means whereby dogs are afforded protection, and has proven efficient in Connecticut. This protection given the dogs also insures the human family to a greater degree of protection than any other methods known.
>
> The statistical evidence of the efficiency of the prophylactic treatment of rabies has been too convincing to permit of doubt by reasoning people, however unconvincing it still remains to some zoöphilic or antimedical sentiments. People with these ideas have been the biggest hindrance in control of rabies in Connecticut.

The Commissioner's office states further:

> We attribute the great reduction in rabies during 1935 and 1936 to the enforcement of rigid quarantine regulations, such enforcements being conducted by special agents of this department, together with protection afforded by vaccination.

In Massachusetts, last year, over 70 clinics were conducted by boards of health, and over 25,000 dogs were vaccinated at these clinics. There has been less rabies in Massachusetts and fewer people have had to take the treatment than in any time during the past since these records have been kept.[14]

The following opinion of a health officer[15] is worthy of consideration:

> In the meantime the Health Officer can well add to his antirabic armamentarium the weapon of dog inoculation. We do not need to wait for the development of a method which will protect every dog against an intra-cerebral inoculation of the most potent virus. As with diphtheria, progress will come as years pass. We are sure today that the method does increase the level of resistance so it is fitting that its use should be further extended. But in so doing, it is well that we should avoid extravagant claims. There is no need for claiming for the method advantages that we do not know

it to possess. Suffice it to say for the moment that it helps to protect the dog. It is far easier to teach the public that the method is more effective than we had realized than it is to disillusion a public whose hopes had been raised to dizzy heights. Nor should we be concerned with the fact that all dogs in the community are not reached. In diphtheria, virtual control is possible if less than half are protected, in spite of the fact that the existence of carriers may add to the menace for the unprotected. In rabies, we are not bothered, as far as we know, by carriers. What figures are now available give us reason to believe that the analogy between the diseases is not too greatly at variance.

In conclusion, the author wishes to reiterate that this is a brief review of the experience and opinions of representative health workers and others on the value of the use of antirabies vaccination in dogs, once yearly, as a part of a public health program in controlling this disease and acting as a greater safeguard to human health. The data presented have been taken from published reports or from personal communications from the authorities quoted.

SUMMARY

1. The real value of a biologic for a certain purpose is shown by carefully evaluated clinical application.

2. To be of value, a vaccine need not be 100 per cent effective, but should be reasonably effective and safe, and its limitations recognized.

3. To control rabies, it is essential that the cycle of propagation of the disease from one animal to another be broken.

4. Field evidence shows that rabies vaccine given to dogs annually as a part of a public health program is of great value in controlling this disease.

REFERENCES

[1]Umeno, S. and Doi, Y.: Kitasato Arch. Exp. Med., iv (1931), p. 89.

[2]Schoening, H. W.: Jour. Agr. Res., xxx (1925), p. 431.

[3]Broerman, A., and Edgington, B. H.: Ohio Agr. Exp. Sta. Tech. Bul. 423 (1928).

[4]Schoening, H. W.: Amer. Jour. Pub. Health, xxvi (1936), pp. 265-267.

[5]Schoening, H. W.: Jour. Agr. Res., xxx (1925), p. 431.

[6]Schoening, H. W.: Corn. Vet., xviii (1928), pp. 105-111.

[7]Schoening, H. W.: Jour. A. V. M. A., lxxvi (1930), n. s. 29 (1), pp. 25-33.

[8]Kando, S.: Proc. 5th Pacific Sci. Cong., 1933, pp. 3055-3060. Abst. in Vet. Bul. vi (1936), p. 132.

[9]Martin, W. D. (Health Officer, Albany, Ga.): Personal communication.

[10]Norton, R. (Health Officer, Rocky Mount, N. C.): Trans. Southern Branch Amer. Pub. Health Asso., 1935.

[11]Kitchen, F E. (Dept. of Health, Greenville, S. C.): Personal communication.

[12]Denison, G. A. (Birmingham, Ala.): Trans. Southern Branch Amer. Pub. Health Assn., 1935.

[13]Schorer, E. H. (Health Director, Kansas City, Mo.): Personal communication.

[14]Jakeman, H. W. (Boston, Mass.): Personal communication.

[15]Anderson, G. W.: Jour. A. V. M. A., lxxxvi (1935), n. s. 39 (1), pp. 76-83.

THE TREATMENT OF BOVINE MASTITIS WITH SULFANILAMIDE

A Preliminary Report*

By A. J. ALLOTT, *Newburgh, N. Y.*

INTRODUCTION

Following the observations of Domagk[1] that certain azo dyes containing the benzene sulfonamide nucleus presented extraordinary curative power in animals experimentally infected with virulent streptococci, investigations in France, England in this country confirmed these.

While abundant evidence showed that the azo benzene sulfonamide dyes were effective, it was determined by a group of French investigators,[2] working independently, that the essential nucleus in all of these was para-aminobenzensulfonamide or "sulfanilamide," which is the name accepted by the American Medical Association for this chemical compound.†

In the United States, Long and Bliss,[3] of Johns Hopkins, were the first to report a series of cases of human beings treated with sulfanilamide and its derivatives. These were all individuals suffering from infections with beta-hemolytic streptococci. For the first time in the history of medicine, an agent of relatively simple chemical structure was found to possess chemotherapeutic powers against bacteria.

Although the group of hemolytic streptococci causing bovine mastitis is usually not pathogenic for man, the infections in cattle cause tremendous financial loss every year. The cases reported in this paper were selected chiefly because the prognosis was practically hopeless. Consequently, even though the series is small and the improvement not so great as might be desired, it is hoped that the encouraging results obtained in such hopeless situations will stimulate the use of this drug in early acute cases.

In the latter, where the infection has not yet produced tissue damage, it is possible that subsidence of the infection with a minimum of scarring and destruction of glandular tissue will be obtained.

From the experimental work of Marshall, Emerson and Cutting,[4] correlated with the clinical observations of Long and Bliss, it was found that sulfanilamide is rapidly absorbed from the gastro-intestinal tract, and to obtain optimum therapeutic results

*Received for publication, August 26, 1937.

†Sulfanilamide used in this investigation was supplied through the courtesy of Merck & Co. Inc.

it was necessary to administer frequent doses to maintain the desired concentration in the blood.

With the exception of a short report in the *New England Journal of Medicine*,[5] reporting the successful treatment of a case of bovine mastitis caused by the hemolytic streptococcus, together with two other similar cases communicated to the author, there were no published data on dosage in the cow. These reports were not then known to the writer of this paper, and consequently various amounts of the drug were tried in the cases treated.

Case Reports

The following case reports, together with clinical and laboratory data are presented:

Case 1: Guernsey cow, normal in appearance, temperature, appetite and condition. Chronic mastitis found in the four quarters of the udder; typical class 4 udder. The right front quarter was the only one secreting milk normal in appearance, but it was positive to the thybromol test. There were medium indurations in the right hind quarter and both left quarters had large indurations. The milk from these quarters was very greatly changed, varying from the usual thick curds to the watery type milk. Laboratory examinations were made of each sample of milk from each quarter as shown in table I, giving the results over a period from March 18, 1937, to April 20, 1937.

On March 18, 1937, 30 grams of sulfanilamide was administered twice a day in a capsule. Medication continued until March 31, 1937. Marked improvement in udder and milk was noted for a very short period after the initial dose of sulfanilamide and continued for several days after medication ceased. Laboratory and physical examinations continued until April 20 and May 4, respectively. The peak of improvement, as shown in table I, was reached on March 31, 1937, and continued until April 4. Then the condition gradually returned to its former status. During this period, the animal showed no change in physical appearance and condition. The temperature, appetite and general health remained as at the start of medication. The milk made quite a noticeable change, becoming negative in three quarters as shown by the strip-cup and the physical appearance of the milk, except for the left front quarter which was noticeably changed to a watery type. The udder itself had undergone a change; the left front quarter had atrophied considerably and the indurations in the other quarters were smaller.

From the above findings several important facts were observed. First, that no systemic reaction was evident from medication

TABLE I—*Data on case 1.*

DATE	SAMPLE	METHOD OF EXAMINATION			
		BREED	HOTIS	SMEAR	BLOOD-AGAR
March 19	1	+	++	+	+
	2	+	++	+	+
	3	+	+	+	+
	4	+	++	+	−
March 20	1	+	++	+	+
	2	+	++	+	−
	3	+	?	+	−
	4	+	++	−	−
March 22	1	+	+	+	+
	2	−	+	+	−
	3	−	?	−	−
	4	+	+	+	−
March 25	1	−	++	+	−
	2	−	+	+	−
	3	−	−	−	−
	4	+	+	+	−
March 26	1	−	−		−
	2	−	−		−
	3	−	+	−	−
	4	+	+	−	−
March 27	1	−	+	−	
	2	−	+	−	
	3	−	−	−	
	4	+	++	+	−
March 29	1	−	−	−	−
	2	−	+	−	−
	3	−	−	−	−
	4	+	++	+	−
March 30	1	−	+	−	−
	2	−	+	−	−
	3	−	+	−	−
	4	+	++	+	+
March 31	1	−	−	−	−
	2	−	−	−	−
	3	−	−	−	−
	4	+	++	+	+
April 1	1	−	−	−	
	2	−	−	−	
	3	−	−	−	
	4	+	++	+	−

TABLE I—*Data on case 1—Continued*

DATE	SAMPLE	METHOD OF EXAMINATION			
		BREED	HOTIS	SMEAR	BLOOD-AGAR
April 2	1	−	−	−	
	2	−	−	−	
	3	−	−	−	
	4	+	++	+	+
April 3	1	−	++	+	+
	2	−	−	−	
	3	−	−	−	
	4	+	++	+	+
April 5	1	−	++	+	−
	2	−	++	+	−
	3	−	++	−	−
	4	+	++	+	+
April 8	1	+	++	+	
	2	+	++	+	
	3	+	?	−	
	4	+	++	−	
April 10	1	+	++	+	
	2	+	++	+	
	3	+	++	+	
	4	+	++	+	
April 11	1	+	++	+	
	2	+	++	+	
	3	+	?	+	
	4	+	++	+	+
April 13	1	+	++	+	+
	2	+	++	+	−
	3	+	++	+	+
	4	+	++	+	−
April 14	1	+	++	+	−
	2	+	++	+	−
	3	+	++	−	+
	4	++	++	+	+
April 20	1	+	++	+	+
	2	++	++	+	−
	3	++	++	+	+
	4	+	++	+	+

? = unsatisfactory.

with sulfanilamide in 30-gm doses twice a day. Secondly, that a speedy reduction in all counts and bacteria was noted which lasted for several days following medication. Third, a definite improvement was noted in an udder which was beyond repair.

TABLE II—*Data on case 2.*

DATE	QUARTER	HOTIS	STREPTOCOCCI	CELL COUNT
June 4	R. R.	+	+	9,600,000
June 10	R. R.	−	−	3,600,000
June 11	R. R.	−	−	9,000,000
June 13	R. R.	−	−	2,400,000
June 14	R. R.	+	+	1,200,000
June 15	R. R.	−	−	1,200,000
June 16	R. R.	−	−	1,600,000

TABLE IIa—*Data on case 2.*

DATE	SAMPLE	METHOD OF EXAMINATION		
		BREED	HOTIS	SMEAR
May 28	1	−	−	−
	2	+	+	+
	3	−	−	−
	4	−	−	−
June 4	1	+	++	+
June 10	59	−	−	−
	60	+	−	−
	58	−	−	−
	55	−	−	−
June 11	53	+	−	−
	54	−	−	−
	56	−	−	−
	57	−	−	−
June 13	6	−	−	−
	7	+	−	−
	9	−	−	−
	11	−	−	−
June 14	1	+	−	−
	2	−	++	+
	3	−	−	−
	4	−	−	−
June 15	5	+	−	−
	6	−	−	−
	7	−	−	−
	8	−	++	+
June 18	9	+	−	−
	10	−	−	−
	11	−	−	−
	12	−	−	−

Case 2: A large Holstein cow, with a class 3 udder, showing chronic mastitis with deep-seated indurations in the right rear quarter. She gave marked reactions to the thybromol test and the strip-cup showed chunks and watery milk. The three other quarters were normal to the thybromol and strip-cup, and there were no indurations in these glands. Laboratory examinations were made similar to case 1 and as charted, to mark the findings.

On June 4, 1937, 30-gm twice a day was given. No marked changes were noticed on laboratory or physical examinations and on June 7, 1937, 60-gm twice a day was given and continued until June 10. During this period, no systemic reaction to medication was noticed. The laboratory findings showed improvement, the milk became negative to the thybromol test and there were no findings on the strip-cup. Sulfanilamide was discontinued from June 10 to June 15, during which time the condition lapsed somewhat to its former state. Medication was resumed at 60-gm twice a day to June 18, when the milk become normal in appearance, the strip-cup findings were negative, as well as the thybromol test. From June 18 to July 12, no medication was administered and the findings lapsed slightly to the former state, but the physical appearance of the milk was much improved as was the condition of the quarter of the udder. No decrease of the milk supply was shown during the treatment.

The sample findings as in case 1 are evident and also that marked improvement was found during administration of sulfanilamide.

Case 3: A large Holstein cow, chronic mastitis with type 3 upper, right front quarter showing indurations throughout quarter, watery type milk and positive to the thybromol. June 5, 60-gm twice a day until June 10, discontinued for one day and then continued from June 11 to June 16. From June 10 to June 15 the milk from this quarter became normal in appearance, negative to strip-cup and thybromol test and laboratory findings. From June 15 to July 9, no medications were used and udder lapsed to near its former state.

While this cow was being treated very marked improvement was noted, but this decreased slowly from the time of the termination of the treatment. The results show the definite influence of sulfanilamide.

Laboratory Data

The examinations consisted of the following tests:

1. The direct microscopic examination of the sample as received, by the Breed method.

TABLE III—*Data on case 3.*

DATE	SAMPLE	METHOD OF EXAMINATION		
		BREED	HOTIS	SMEAR
June 5	2	+	++	+
	3	+	++	−
June 10	4	−	−	−
June 11	5	−	−	−
June 17	1	+	−	−

TABLE IIIa—*Data on case 3.*

DATE	QUARTER	HOTIS	STREPTOCOCCI	CELL COUNT
June 5	R. F.	+	+	2,000,000
June 7	R. F.	+	−	960,000
June 10	R. F.	−	−	250,000
June 11	R. F.	−	−	500,000
June 17	R. F.	−	−	12,000,000

2. Incubation of the sample for 24 hours with brom-cresol-purple (0.5 cc of a 0.5 per cent sterile aqueous solution of the dye to 9.5 cc of sample), according to the method of Hotis. Tubes exhibiting a color change or yellow flakes after incubation for 24 hours at 37° C. were examined microscopically for the presence of streptococci.

3. A portion of the same as received was reserved and incubated for 24 hours at 37° C., after which a loopful was streaked on blood-agar plates in order to determine if the organisms were hemolytic. (This was discontinued later in the test, since reactions were so slight.)

In the tabulation of results, the following scheme is used:

Case 1: A negative (—) sign in the column marked "Breed" indicates that the milk appeared normal under the microscope. A plus (+) sign indicates that a cell count of over 1,000,000 per cc was found. The plus (+ +) signs indicate that streptococci associated with leukocytes were found.

Under the column marked "Hotis" a single plus (+) sign indicates that a color change only was observed. Two plus (++) signs indicate that a color change and yellow flakes were found. A negative (—) sign indicates that neither color change nor yellow flakes were observed.

Under the column marked "Smear" a plus (+) sign indicates that streptococci were observed in the microscopic preparation from the Hotis tube. A negative (—) sign indicates that no streptococci were found in the preparation.

Under the column marked "Blood-Agar" a plus (+) sign indicates that the organism produced hemolysis. A negative (—) sign indicates that no growth was noted on the blood-agar plate.

Cases 2 and 3: A negative (—) sign in the column marked "Breed" indicates that a cell count of less than 500,000 per cc was found. A plus (+) sign in this column indicates that a cell count of over 500,000 was found.

A single plus (+) sign in the column marked "Hotis" indicates that a color change was noted. A double plus (+ +) sign in this column indicates that a color change and yellow flakes were observed.

A plus (+) sign in the column marked "Smear" indicates that long-chain streptococci were demonstrated in microscopic preparations made from the Hotis tube.

The blood-agar plates were not included, for the reason that in the two previous cases only relatively few plates gave positive results. It was felt that, with this type of sample, while a positive reaction on blood-agar was of significance, a negative test was not necessarily conclusive.

Discussion

From these observations it seems to be clear that in the dosages employed there has been a definite response, with both clinical and laboratory evidence of improvement. In the human being the dose has been recommended as 1-gm per 9.1 kilograms (20 pounds) of body weight. Roughly, this is the dose that has been given to these animals. For the larger breeds, 30-gm a day in four divided doses may be advantageous. It is known from the work that Marshall and his co-workers referred to above, that even spacing of doses at about four-hour intervals is most satisfactory. In veterinary practice this frequency may be inconvenient, but it is believed that a single dose or even two doses per day may not maintain the optimal constant concentration in the blood. We recommend, therefore, 30-gm four times a day.

The results of this work with chronic infections would seem to indicate that early and thorough treatment of acute infections, before necrosis, abscess formation, and scarring have occurred, would offer considerably more hope for a successful termination of the disease process. Work on early acute cases is now under way and the results will be reported in the near future.

Conclusions

1. Three cases of bovine mastitis due to hemolytic streptococci and treated orally with sulfanilamide are reported.

2. Sulfanilamide is an effective chemotherapeutic agent against this otherwise hopeless condition.

3. The dosage has been discussed with the reservation in mind that this may require revision in the light of more extensive experience.

Acknowledgment

The author wishes to express his appreciation to John F. Kingsley, Ph. Ch., of Newburgh, New York, who performed the bacteriological tests recorded in this paper.

References

[1]Domagk, G.: Deut. Med. Wchnschr., lxi (1935), 2, pp. 250-253.
[2]Trefouel, J., Nitti, F., and Bovet, D.: Compt. Rend. Soc. Biol., cxx (1935), p. 956.
[3]Long, P. H., and Bliss, E. A.: Jour. Amer. Med. Asso., cviii (1937), pp. 32-37.
[4]Marshall, E. K., Jr., Emerson, K., Jr., and Cutting, W. C.: Jour. Amer. Med. Asso., cviii (1937), pp. 953-957.
[5]Stevens, N. C.: New Eng. Jour. Med., ccxvi (1937), 6, p. 1091.

New York Now Tuberculosis-Free

Approximately 13 years after the first county (Essex) in the state of New York was accredited, the entire Empire State was designated by the U. S. Department of Agriculture as a modified accredited area. This was on October 1, 1937, and marked the cleaning up of all territory east of the Mississippi River.

New York was the 46th state to be accredited. California and South Dakota are now the only states in which there is any non-accredited territory. The accrediting of New York is to be celebrated in Albany, on November 4, with appropriate ceremonies.

Michigan Couples Celebrate Anniversaries

Dr. and Mrs. Elijah E. Patterson, of Detroit, Mich., observed their golden wedding anniversary on October 19. Both were born in the Motor City and have lived there all their lives. Dr. Patterson has practiced in Detroit for about 40 years.

Dr. and Mrs. Otto W. Schubel, of Quincy, Mich., were pleasantly surprised on October 23, when a group of friends and relatives called at their home to help celebrate their silver wedding anniversary. Dr. Schubel has practiced in Quincy for nearly 25 years.

VETERINARY CONTRIBUTIONS TO HUMAN HEALTH*

By JOHN R. MOHLER, *Washington, D. C.*
Chief, Bureau of Animal Industry
United States Department of Agriculture

Health is so valuable a possession that most of us use every recourse at our command to guard it and to recover it, if lost. Even back in primitive times, a keen interest in health was evident from the efforts of tribal medicine men to cure human ills. In their ignorance they devised many concoctions obtained from many sources. Some of the ingredients were herbs. Others were of animal origin. With increasing enlightenment achieved through research, man gradually discarded unsound beliefs and build up a vast supply of reliable medical information.

Yet the medical profession very wisely continues to augment its own knowledge and resources with experience gained in related fields. One of the most fruitful is that of veterinary science, which deals with the functions and health of animals.

Medical and veterinary science have always had parallel objectives. The former contributes to human health directly; the latter does so indirectly. For example, nearly half a century ago, three research workers of the Bureau of Animal Industry sought a means of controlling tick fever in cattle. These men, Smith, Kilbourne and Curtice, discovered that the cause of tick fever was a protozoan parasite which, in turn, was carried by another parasite, the cattle tick. The tick served as an intermediate host. This discovery blazed the trail to new fields of veterinary and medical knowledge. It led to the discovery that the infection of many dangerous human diseases is carried in a similar manner. These diseases include yellow fever, malaria, typhus fever, Rocky Mountain spotted fever, and others. Knowledge of the cause and mode of transmittal is, of course, the basis of effective prevention and control.

More recently, veterinary science has contributed to human health through a great reduction in bovine tuberculosis. The bovine type of tubercle germ is especially dangerous to children. It tends to cause, among other things, bone and joint involvement, sometimes developing into the condition known as "hump-

*Radio address delivered during the 66th annual meeting of the American Public Health Association, New York, N. Y., October 5-8, 1937, on invitation of Dr. Reginald M. Atwater, Committee on Publicity.

back." With the steady decrease in bovine tuberculosis, the number of such cases has greatly decreased.

Bovine tuberculosis is now rapidly approaching complete eradication. Through the systematic testing of cattle and the removal of tuberculous animals, the average extent of bovine tuberculosis is now only about 0.4 of 1 per cent, in contrast to nearly 5 per cent some 20 years ago, when systematic eradication work began. In that time, veterinary inspectors have detected more than 3,500,000 tuberculous cattle and removed them for slaughter. This vast reduction in tuberculous cattle and the undertaking in general have contributed to human health by reducing a former source of infection.

Another disease enemy common to man and certain lower animals, including hogs, is trichinosis. It is caused by a parasite of microscopic size. A small percentage of hogs acquire this parasite by eating products in which the parasite is present. Veterinary research has shown that hogs fed on raw garbage are especially likely to pick up the infection. However, thorough cooking of pork is a positive means of prevention. Prolonged refrigeration at low temperatures likewise is an effective safeguard. Various modern methods of processing those pork products that are commonly eaten with little or no cooking are further means of protecting persons from trichinae. In addition to these precautions, a system of swine sanitation, developed through veterinary research, has proved to be effective in stopping the life-cycle of infection of the trichina parasite, with resulting benefit to animal and human life.

As other typical veterinary contributions to human medicine we have the class of drugs known as anthelmintics. Carbon tetrachloride is one of those. Its successful use in removing hookworms from dogs suggested its possible value in serving the same purpose for human beings. Tests soon showed the soundness of this belief. A similar drug, tetrachlorethylene, was first used on dogs and later for human beings. Many biological products and glandular extracts used in human medicine are derived from animals slaughtered under federal inspection. It is highly important that these products be obtained from a safe source and veterinary science provides this necessary measure of safety.

Veterinarians, of course, have been greatly interested in the control of rabies. Many warm-blooded animals are susceptible to this disease, but dogs in particular need close observation because of their roving habits. The veterinary profession has sought to aid health officers and others in obtaining adequate regulations and related means of protecting society from rabies.

These remarks would be incomplete without at least a brief mention of the extensive federal meat-inspection service now conducted in some 250 cities and towns in the United States. Although little publicized, this service protects the well-being of almost every person in the United States. The service includes the veterinary examination of about 70,000,000 animals annually. The inspection extends, of course, to the resulting meat and many meat food products. The public can readily identify federally inspected meat products by the familiar circular stamp on fresh meat and labels on canned and packaged goods.

These examples are typical of veterinary service that have been useful to man. In conclusion, I confidently trust that veterinary contributions thus far made to human health are the forerunners of many more to come.

Dr. W. E. Cotton Retires

Dr. William E. Cotton has retired from the employ of the U. S. Department of Agriculture after 44 years of continuous service in the Bureau of Animal Industry. During this period Dr. Cotton was engaged in various lines of veterinary activity and, at the time of his retirement, he was superintendent of the Animal Disease Station of the B. A. I. This was formerly located at Bethesda, Md., but in 1935 was moved to Beltsville, Md., where it became a part of the National Agricultural Research Center.

Dr. Cotton is best known for his research work with a number of animal diseases, including Bang's disease, tuberculosis, foot-and-mouth disease, tick fever, and other maladies. He is the author of many scientific articles and delivered one of the principal addresses at the Twelfth International Veterinary Congress, in New York, in 1934. Dr. Cotton has accepted a position on the teaching staff of the Alabama Polytechnic Institute, at Auburn.

Four Veterinarians Suffer from Undulant Fever

The following veterinarians have been reported as recently having suffered from attacks of undulant fever: Dr. John P. Turner (U. P. '90) and Dr. Ervin A. Eichhorn (U. P. '35), of Washington, D. C.; Dr. C. L. Everson (O. S. U. '20), of Baltimore, Md., and Dr. James W. Sullivan (U. S. C. V. S. '25), of Hagerstown, Md.

STOMATITIS IN YOUNG LAMBS INVOLVING ACTINOMYCES NECROPHORUS AND THE VIRUS OF CONTAGIOUS ECTHYMA*

By H. MARSH *and* E. A. TUNNICLIFF
Montana Veterinary Research Laboratory†
Bozeman, Montana

This report records observations on an outbreak of stomatitis in lambs three to four weeks old, in which the primary etiological agent was the virus of contagious ecthyma, commonly known as sore-mouth. The principal lesions in this outbreak were on the buccal mucous membrane, with only a small percentage of the lambs showing skin lesions on the lips. Boughton and Hardy[1] state that sore mouth may occur in very young lambs, but the same authors[2] also say that lesions on the buccal mucous membrane are rare in their experience. In Montana, sore mouth occurs in lambs four to six months old, but we have never before known of its occurrence in very young lambs.

The cases reported here occurred in May, 1936, on a ranch where there were about 3,000 lambs. The owner first noticed the condition on May 8, when lambs about two to three weeks old were being docked. Observations were made on May 12, when one band of the lambs was seen on the range. On May 16, this band, consisting of 930 lambs, was corralled and every lamb examined. About 450 showed lesions to a greater or less extent, of which 50 to 75 were advanced cases. The origin of the lesion was in the gums at the base of the incisor teeth. The gum was reddened and spongy, and in many cases the exuberant granulation tissue completely covered the teeth. On the surface of the lesion there was a gray or white necrotic zone characteristic of *Actinomyces necrophorus*‡ infection. Smaller necrotic lesions occurred

*Contribution from Montana State College, Agricultural Experiment Station. Paper No. 97, Journal Series. Received for publication, July 30, 1937.
†Montana Experiment Station and Montana Livestock Sanitary Board coöperating.
‡We consider that this organism should not be included in the genus *Actinomyces*, but we use this name because we have adopted Bergey's classification as standard

also on the dental pad, the roof of the mouth, and the buccal surface of the lips. The characteristic odor of *A. necrophorus* lesions was present. A small percentage of the lambs also showed characteristic sore-mouth scabs on the lips. In many lambs the buccal lesions were so extensive that eating grass was impossible, but nursing was not interfered with to any extent, and the most of the lambs were well nourished.

FIG. 1. Lesions on gum and lip produced by inoculation with unfiltered material from field case.

The original diagnosis was necrobacillosis, and treatment was advised to prevent the extension of the infection. The exuberant tissue was removed by treating with silver nitrate sticks and rubbing off with a cloth. The small necrotic lesions were cauterized with silver nitrate. The mouths were then swabbed with a 10 per cent potassium permanganate solution. The treated lambs were examined on May 20, when it was found that the lesions were much less extensive than before treatment. Apparently no new cases were developing. The owner reported that the condition had not become serious in two other bands of lambs in which no treatment had been used.

When it was found that over 50 per cent of the lambs in one band were affected, it seemed unlikely that the original diagnosis of necrobacillosis as the primary condition could be correct. Therefore, material was brought to the laboratory to be examined for the sore-mouth virus.

One of the specimens collected consisted of the granulation tissue removed from the gum at the base of the incisor teeth. Smears from this material showed the presence of *A. necrophorus* in large

FIG. 2. Lesion on lips produced by inoculation with filtrate. The gum also was inoculated, but no lesions developed.

numbers. The organism was recovered from a rabbit inoculated with this material.

A suspension in water was prepared by grinding 1.25 gm of the tissue with sand in 10 cc of distilled water. This suspension was held at 4° C. for three days. The suspension was then digested by adding 1 cc each of liquor pancreatin and 1 per cent sodium carbonate solution and holding for five hours at 37° C. This digested material was added to a buffered saline solution of pH 7.6, in the ratio of 1.4 cc 40 cc. The fluid was clarified by centrifugation in pyrex tubes, after which the reaction was 7.3.

The fluid was then passed through a Berkefeld V filter, after which it was bacteria-free, as determined by inoculation into culture media.

The suspension before filtration and the filtrate were tested for the presence of virus by inoculation into lambs. Two sore-mouth-immune lambs and two unvaccinated lambs were inoculated with the unfiltered suspension, and two unvaccinated lambs were inocu-

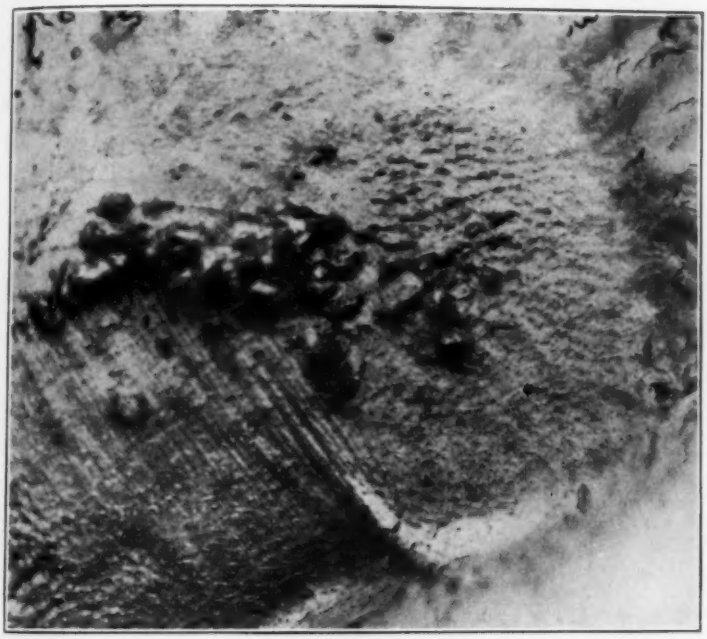


FIG. 3. Inguinal lesions produced by inoculation with filtrate.

lated with the filtrate. The immune lambs showed a very slight reaction to the inoculation of the suspension, while the two susceptible lambs developed well-marked lesions. The filtrate produced no lesions in one lamb, but slight lesions of sore mouth developed on the lip of the other lamb.

Two filtrates were prepared, by the method described above, from lip scabs collected from one of the field cases. These filtrates did not produce lesions in inoculated lambs, but the suspensions before filtration produced typical sore-mouth lesions on the lips, and also produced gum lesions similar to those seen in the field cases. Figure 1 shows both the lip lesion and the lesion on the buccal mucosa.

The lip scabs were dried and stored in the refrigerator for one year and were then tested again for the presence of the filtrable virus of sore mouth. The method of preparation of the filtrate differed from that used when the material was fresh. Two-tenths of a gram of the dried scab was triturated with 10 cc of distilled water. The suspension was frozen in a pyrex tube and held at freezing temperature over night. Ten cc of the suspension was

FIG. 4. Spontaneous stomatitis developing from accidental exposure to the virus.

then mixed with 30 cc of hormone broth and 25 cc of sheep serum, centrifuged to clear, and passed through a Berkefeld N filter. Lamb 109, 36 days old, and lamb 113, 33 days old, were inoculated with this filtrate. The filtrate was swabbed over lightly scarified areas on the lips, the gum at the base of the incisors, and the skin of the left inguinal area. Both lambs developed typical sore-mouth lesions on the lips and the inguinal area, but no lesions on the gums. Figures 2 and 3 show the lip and inguinal lesions of lamb 113. This result definitely established the presence of the filterable virus of contagious ecthyma.

A spontaneous case of stomatitis of the same nature as that observed in the field outbreak occurred in a lamb born at the lab-

oratory. This lamb was born on July 15, and on August 3 was found to have developed the condition shown in figure 4. This photograph shows the same type of lesion as seen in the milder cases in the field outbreak. There was a proliferation of tissue at the base of the incisor teeth, having the red granular appearance of granulation tissue. There were also two small growths of papillomatous appearance on the buccal surface of the upper lip at the base of the gum. Slight sore-mouth lesions developed at the commissures of the lips.

The only possible source of the infection in this case was exposure in a truck which had been used to remove bedding from the pens in which the experimental cases were produced. This lamb was brought in from the pasture in the truck a few days after birth.

Discussion

This outbreak of stomatitis is of interest because of the early age at which the disease appeared in the lambs, and because both the virus of contagious ecthyma and a strain of *A. necrophorus* of relatively low virulence were involved. Apparently the lesions on the mucosa of the mouth developed as a result of the activity of both *A. necrophorus* and the contagious ecthyma virus. It was possible to reproduce the lesions on the buccal mucosa of lambs experimentally by the inoculation of the unfiltered suspension of material from the mouth lesions, but the filtrates failed to produce lesions on the mucosa, although they produced characteristic sore-mouth lesions on the lips. Strains of *A. necrophorus* of relatively low invasive power, and with morphological and cultural characteristics similar to those of the strain isolated in this outbreak, have been found in buccal lesions in other cases of virus sore mouth studied in this laboratory. The probable significance of *A. necrophorus* found in such cases will be reported later in another paper.

References

[1]Boughton, I. B., and Hardy, W. T.: Immunization of sheep and goats against sore mouth (contagious ecthyma). Texas Agr. Exp. Sta. Bul. 504 (1935).

[2]Boughton, I. B., and Hardy, W. T.: Contagious ecthyma (sore mouth) of sheep and goats. Jour. A. V. M. A., lxxxv (1934), n. s 38 (2), pp 150-178.

Beavers in Pennsylvania

It was recently reported in *Science* that 96 beavers, imported between 1917 and 1924 into Pennsylvania, where they had been extinct for 70 years, have multiplied to over 15,000, according to the latest beaver count.

CONGENITAL DEFORMITY OF UTERUS AND URINARY BLADDER IN A FEMALE MINK*

By EARL F. GRAVES, *Poynette, Wis.*
Wisconsin State Fur Farm

The subject was a matured, female mink which had accepted the service of a male two weeks before death. Externally the animal appeared perfectly normal. Upon autopsy, it was noted that there was an unusual uterine, bladder arrangement.

There was a normal-appearing left ovary but no left horn at all, just the broad ligament, perfectly devoid of any uterine horn. There was a normal-appearing right ovary with uterine horn suspended in the broad ligament, but it ended at what normally would have been the body of the uterus. In place of the uterine body was the urinary bladder, full of urine. There was a fold of tissue in the neck that apparently acted as a valve.

Thus we have two normal-appearing ovaries, one uterine horn, and no uterine body, but in its place a urinary bladder. The one horn was simply a closed tube and did not communicate with the bladder but was firmly fastened to it as a part of it.

Rodents

Rodentia is the largest order of mammals and consists of about 12,000 species. The order Rodentia includes squirrels, mice, rats, prairie-dogs, gophers, beavers and many other groups. Scattered throughout the United States are over 40 species of squirrels and chipmunks. These rodents are influenced by their environment. Their food is that which grows in the vicinity of their habitat. The increase or decrease in the number of rodents may depend somewhat upon their natural enemies as well as the supply of food.

Hornets' Federation

Sam, the colored driver of a mule team, saw a lizard crawling up a tree. He flourished his long whip and very deftly snapped off the lizard's head. Farther along the road, with skillful precision, he picked a horsefly off the fence with the same weapon. A white passenger in the coach finally said, "Sam take a crack at that hornets' nest there.' Sam grinned and shook his woolly head, "No suh, them fellows is organized."

—*Selected.*

*Received for publication, April 17, 1937.

STUDIES ON BOVINE MASTITIS. XII. Mastitis due to staphylococci. F. C. Minett. Jour. Comp. Path. & Therap., 1 (1937), p. 101.

Cases of mastitis, acute and chronic, caused by pathogenic staphylococci (*Staphylococcus aureus*) have been found to be of such a nature as to be confused with mastitis caused by streptococci. In its commonest form the disease is chronic, the stroma being ultimately involved. Enduring infection leads to diffuse induration of the gland with some decrease in milk yield and qualitative changes in the milk, such as excessive alkalinity, increase in leukocytes, a lowered specific gravity and solids-not-fat content. In distinguishing pathogenic staphylococci from nonpathogenic types commonly present in normal udder milk, emphasis is laid upon the importance of the technic adopted. Agar containing sheep or ox blood from young animals is most suitable; the toxins produced by *Staphylococcus aureus* produce characteristic changes on this medium.

ON THE VALUE AND LIMITATIONS OF TISSUE VACCINES AGAINST RINDERPEST. R. L. Cornell and S. A. Evans. Jour. Comp. Path. & Therap., 1 (1937), p. 122.

No marked differences in the antigenic properties of incubated glycerin spleen vaccine and formolized glycerin spleen vaccine were noted when cattle were vaccinated with these products against rinderpest. Both types of vaccine failed to produce solid immunity in all animals with doses up to 3 grams of spleen per 100 kilos body weight. The infectivity of vaccinated animals when reacting to virus and the capacity of vaccinated animals to contract rinderpest by contact are noted. It is suggested that the difference between blocked-out reactions which result in solid and permanent immunity and those which do not is that the virus had established itself and multiplied in the tissues of the animal in the former case, but failed to do so in the latter. Some animals during the period of blocked-out reactions may also be infective

to others. Vaccination should be regarded as a weapon for the control of rinderpest rather than a promising agent for its eradication.

A Study of the Heteroallergic Reactivity of Tuberculin Desensitized Tuberculous Guinea Pigs in Comparison with Tuberculous and Normal Guinea Pigs. Margaret W. Higginbotham. Amer. Jour. Hyg., xxvi (1937), p. 197.

Tuberculous allergic guinea pigs react to intracutaneous inoculations of other bacteria (*Bacillus coli, Staphylococcus aureus, Brucella suis*) with the exaggerated cellular exudation which characterizes the allergic reaction to the tubercle bacillus and its products. Abolition of the specific allergy to *Mycobacterium tuberculosis* in guinea pigs by tuberculin treatment decreases markedly the heteroallergic activity of these animals to *B. coli*, *S. aureus* and *B. suis*. This phenomenon, the presence or abolition of the allergic or heteroallergic reaction, is not observed on the injection of a virulent *Corynebacterium diphtheriae* organism, owing to the marked toxic necrosis in all animals. Broth medium exercises a desensitizing action on both specific and heterologous reactivity which is less than that of the corresponding tuberculin. Desensitized guinea pigs exhibit a relative immunity to reinfection which compares favorably with the immunity of the untreated immune animals. Desensitized guinea pigs do not suffer a loss of antibodies in the blood-serum as indicated by the complement-fixation test with *M. tuberculosis* antigen nor do these antibodies increase with tuberculin treatment.

Studies on the Nature of Immunity to Intestinal Helminths. V. Experiments on the rôle of the skin in parenteral immunity and further experiments on passive immunization in Nippostrongylus infections in rats. Asa C. Chandler. Amer. Jour. Hyg., xxvi (1937), p. 292.

Rats previously infected with Nippostrongylus by skin penetration allow as many larvae to reach the intestines as do rats previously infected by subcutaneous injection or by intestinal implantation, whether the test infections are given there or under the skin. More larvae are found in the intestines of uninfected controls due to failure of many larvae to establish themselves in the intestine after reaching it in rats already harboring intestinal worms. Histological examinations failed to demonstrate increased retention of larvae in skin previously exposed to pene-

tration by Nippostrongylus although leukocytic infiltration and eosinophilia were enhanced. Immunization with live *Ancylostoma caninum* larvae afforded no immunity to Nippostrongylus. Rats repeatedly infected with *A. caninum* by skin penetration showed less skin reaction to a heavy penetration by this worm than did rats repeatedly infected by subcutaneous injection. Rats immunized to *A. caninum* failed to show enhancement of skin reaction to penetration by Nippostrongylus larvae, thus proving the heightened skin reaction to be specific. Rats treated with 6 cc per 100 gm of serum from rats highly immunized by repeated skin penetration by Nippostrongylus showed no effect on the successful migration of larvae to the intestine but showed evidence of a slight effect on establishment of worms in the intestine and on egg production. Rats treated with 6 cc per 100 gm of serum from rats immunized by repeated implantation of worms into the intestine failed to show any effect whatever on successful migration to the intestine, on establishment in the intestine or in egg production.

EFFECTS OF TEMPERATURES ON THE OÖCYSTS OF VARIOUS SPECIES OF EIMERIA. Kwei Chang. Amer. Jour. Hyg., xxvi (1937), p. 337.

Unsporulated oöcysts of *Eimeria nieschulzi, E. miyairii, E. separata, E. tenella, E. arloingi, E. faurei* and *E. ninakohl-yakimovi* from hosts presumably on normal diets do not respond to heat in the same manner when tested under identical laboratory conditions. *E. separata* is the most delicate and *E. arloingi* the most resistant species to heat. At 46° C. it takes 40 times the length of exposure for *E. arloingi* as for *E. separata* in order to effect 50 per cent mortality. Unsporulated oöcysts of *E. nieschulzi* from rats which have been on a vitamin B and G deficient diet for nine days prior to experimental infection are much more easily killed by heat than those from hosts which have been fed the normal diet. The natural mortality, that is, failure of sporulation which is not attributable to heat effects, of oöcysts of *E. nieschulzi* from avitaminized hosts is also considerably higher than that of oöcysts from control hosts on normal diet. The addition of an extra amount of vitamin B and vitamin G to the normal diet of the host does not increase the resistance of the unsporulated oöcysts of *E. nieschulzi* to heat. Starvation of the host does not affect the tolerance of the unsporulated oöcysts to heat.

A Study of the Biochemical Reactions of Strains of Clostridium Tetani Isolated from Street Dust. Eric C. Gillis. Amer. Jour. Hyg., xxvi (1937), p. 401.

Twelve strains of anaerobes with terminal spherical spores were isolated from 63 samples of street dust containing mixed material. The proteolytic and saccharolytic properties of *Clostridium tetani* showed marked variations. Nine of the twelve strains studied produced a spasm-producing toxin neutralized by tetanus antitoxin, while three of the strains failed to produce a demontrable toxin. The strains isolated indicate that *C. tetani* has feeble proteolytic properties and does not ferment the carbohydrates.

A Study of the Blood Picture of Poultry and Its Diagnostic Significance. S. F. Cook. Poultry Sci., xvi (1937), p. 291.

Approximately 300 separate determinations on White Leghorn chickens of different ages showed that the erythrocyte count averages 2,500,000, the hemoglobin value 60 and the leukocyte count 30,000. Up to twelve weeks of age, there was a tendency towards increasing hemoglobin and erythrocyte count. In five groups fed different diets the hemoglobin values for three were uniform, while one was low, probably due to vitamin deficiency, and the fifth was high, apparently as a result of feeding casein. The differential counts showed remarkable uniformity. The leukocyte counts in four groups were practically identical, while those in the fifth group were higher. The blood-clotting time, 4.1 to 5.1 minutes, was uniform in all groups.

A Note on the Incidence of Syngamus Trachea in Turkeys with Observations on Nodules Caused by This Parasite. Everett E. Wehr. Poultry Sci., xvi (1937), p. 331.

The examination of 936 turkey tracheas collected at one market in Washington, D. C., has shown 14.7 per cent infestation with *Syngamus trachea*. Nodules occurring as the result of such infestation are of common occurrence. Each nodule was observed to be the result of the attachment to the wall of the trachea of the head of the male worm which in many cases was deeply embedded in the nodular tissue. The male worm may remain attached during most if not all of its parasitic life in the trachea of the turkey and may serve as an anchor for the female worm, which apparently attaches and detaches periodically in order to obtain food.

STUDIES IN COCCIDIOSIS IN CHICKENS. CALCIUM CARBONATE ADDITIONS AND COCCIDIA. C. E. Holmes, C. A. Herrick and G. L. Ott. Poultry Sci., xvi (1937), p. 335.

In two trials more deaths occurred after infection with coccidiosis when 6 per cent of chick-size oyster shell was added to a basal ration containing 0.8714 and 0.9343 per cent calcium and phosphorus, respectively. With higher levels of calcium carbonate, three trials indicated more deaths occurred after infection with coccidiosis. Attempts to determine the effects of different levels of calcium carbonate on mortality from coccidiosis holding the calcium-phosphorus ratio constant were not successful, due to the unpalatability of the ration.

THE PATHOGENICITY TO ANIMALS OF VIRUSES ISOLATED FROM CASES OF HUMAN INFLUENZA. J. McIntosh and F. R. Selbie. Brit. Jour. Exp. Path., xviii (1937), p. 334.

Two strains of virus isolated from the lungs of cases of human influenza were found capable of causing an experimental disease with lung lesions in animals. The lesions were produced in ferrets, mice, rabbits, guinea pigs and a monkey by intranasal inoculation under anesthesia. Large areas of collapse and hemorrhage in the lungs were observed in animals in which the inoculations were fatal. Interstitial pneumonia, characterized by proliferative changes in which large numbers of mononuclear cells predominated, were noted. There was an increase in mononuclear cells of the blood.

IMMUNOLOGICAL RELATIONSHIP BETWEEN THE SWINE AND HUMAN INFLUENZA VIRUSES OF SWINE. Richard E. Shope. Jour. Exp. Med., lxvi (1937), p. 151.

Swine recovered from infection with swine influenza or swine influenza virus alone are usually immune to infection with a mixture of human influenza virus and *Hemophilus influenzae suis*. This cross-immunity is not associated with the presence of demonstrable neutralizing antibodies for the human virus in the sera of the immune animals. Swine recovered from human influenza virus alone are usually not immune to swine influenza. The cross-immunity shown by swine recovered from infection with the viruses of human and swine influenza, respectively, may be due to different mechanisms.

THE EFFECT OF HEMOPHILUS INFLUENZAE SUIS VACCINES OF SWINE INFLUENZA. Richard E. Shope. Jour. Exp. Med., lxvi (1937), p. 169.

Hemophilus influenzae suis, given intramuscularly to swine, elicits an immune response capable of modifying the course of a later swine influenza infection. Heat-killed vaccine appears to be at least as effective as a living one, as judged from clinical and postmortem findings. *H. influenzae suis* was more often completely suppressed in the pneumonic lungs of animals vaccinated with live cultures than in the lungs of those that had received the heated vaccine. The partial protection afforded swine by the bacterial vaccines is of no immediate value, since it has been shown that complete protection to the disease can be gained by means of swine influenza virus vaccines. A partial immunity to the bacterial component of the etiologic complex can be established.

He who has truth at his heart need never fear the want of persuasion on his tongue.—*John Ruskin.*

"SPEYER HOSPITAL! AND DRIVE LIKE MAD!"

—*Courtesy The New Yorker.*

PUBLICATIONS RECEIVED

Further Studies of Diseases Affecting Moose. II. R. Fenstermacher. Reprint from *Corn. Vet.*, xxvii (1937), 1, pp. 25-37.

A Note on the Occurrence of *Linguatula Serrata* (Frohlich, 1789) in Australia. E. M. Pullar. Reprint from *Australian Vet. Jour.*, Apr., 1936, pp. 61-64.

Cysteine-Gelatin as a Differential Medium for *Salmonella Pullorum* and *Salmonella Gallinarum*. W. R. Hinshaw and L. F. Rettger. Reprint from *Proc. Soc. Exp. Biol. & Med.*, xxxv (1936), pp. 44-46.

Biology and Control of the Large Roundworm of Fowls, *Ascaridia Galli* (Schrank 1788) Freeborn 1923, Studies on the. (Bul. 2. Queensland Dept. Agr. & Stock, The Animal Health Sta., Jan., 1937. pp. 106. Illus.)

Swine Erysipelas. G. L. Dunlap and Robert Graham. (Circ. 471. Ill. Agr. Exp. Sta., Apr., 1937. pp. 13. Illus.)

Melophagus Ovinus (Linnaeus, 1758) Infesting a Calf. With a note on the body length of the parasite. E. M. Pullar. Reprint from *Australian Vet. Jour.*, 1937, pp. 72-74.

Pullorum Disease in Ducklings. W. R. Hinshaw and H. A. Hoffman. Reprint from *Poul. Sci.*, xvi (1937), 3, pp. 189-193.

The Incidence of Chagas' Disease in Panama as Determined by the Complement-Fixation Test. Carl M. Johnson and R. A. Kelser. Reprint from *Amer. Jour. Trop. Med.*, xvii (1937), 3, pp. 385-392.

Washington, 1937, Bulletin of the State College of. (Pullman,, Wash., 1937. p. 344.)

Some Investigations on the Virulence and Growth of the B. C. G. Bacillus and Other Tubercle Bacilli. Halfdan Holth. Reprint from a bulletin published in Oslo, Denmark, 1937. pp. 41.

Transmission of Fowl Leukosis. E. P. Johnson. Reprint from *Poultry Sci.*, xvi (1937), 4, pp. 255-260.

Department of Agriculture, New South Wales. Live Stock Diseases Report (No. 12), 1935-1936. Max Henry. (Sydney, N. S. W., 1937. pp. 24.)

Kenya Colony and Protectorate. Department of Agriculture Annual Report, 1936. Volumes I & II. (Nairobi, Kenya, 1937, pp. 184. Price 2/50 per vol.)

Studies on *Corynebacterium Pyogenes*, with Special Reference to Toxin Production. Reginald Lovell. Reprint from *Jour. Path & Bact.*, xiv (1937), 1, pp. 339-355.

Applied Endocrinology in Zoötechnic. Arcadio C. Gonzaga. Reprint *Philippine Jour. Ani. Ind.*, iv (1937), 3, pp. 213-223.

Corneal Opacity in a Dog Treated with Cow's Milk. Arcadio C. Gonzaga. Reprint from *Philippine Jour. Ani. Ind.*, iii (1936), 6, pp. 477-478.

Studies on Bovine Mastitis. XII. Mastitis Due to Staphylococci. F. C. Minett. Reprint from *Jour. Comp. Path. & Therap.*, 1 (1937), part 2, pp. 101-121.

Bovine Mastitis. I. The Significance of the Dose Factor in the Production of Experimental Mastitis. Ralph B. Little. Reprint from *Corn. Vet.*, xxvii (1937), 3, pp. 297-308.

Bovine Mastitis. II. The Production of Mastitis by the Suction of Streptococci into the Duct of the Teat. Ralph B. Little. Reprint from *Corn. Vet.*, xxvii (1937), 3, pp. 309-316.

Report of Extension Veterinarians' Conference

A conference of extension veterinarians was held in connection with the annual meeting of the American Veterinary Medical Association, at Omaha, Nebraska, on Wednesday afternoon, August 18, 1937. Nine different states and the United States Bureau of Animal Industry were represented at this conference. Those in attendance were as follows:

Dr. B. A. Beach, Madison, Wisconsin.
Dr. G. R. Dunlap, Urbana, Illinois.
Mr. W. W. Derrick, Lincoln, Nebraska.
Dr. C. D. Lowe, Washington, D. C.
Dr. J. W. Lumb, Manhattan, Kansas.
Dr. W. T. Oglesby, Baton Rouge, Louisiana.
Dr. E. N. Stout, Fort Collins, Colorado.
Dr. K. W. Stouder, Ames, Iowa.
Dr. Gilbert S. Weaver, Brookings, South Dakota.

A round-table discussion, introduced by Dr. C. D. Lowe, was the main feature of the meeting. Dr. K. W. Stouder introduced the subject of avian tuberculosis as being one which demands serious attention in his state. He discussed the insidious nature of this disease and the amount of loss it was causing in poultry flocks and swine herds. Measures of control and prevention were discussed by Doctors Beach and Lowe and others in attendance. The possibility of holding poultry schools and clinics in coöperation with the practicing veterinarians and county agents of a county was proposed; the idea being to assist in making poultry-owners more aware of the fact that veterinarians located in their communities can be of great assistance in continuously helping them to plan disease control and prevention for their flocks.

It was brought out that since in most states no funds are available for control work on avian tuberculosis, efforts should be made to interest flock-owners in using their local veterinarian in assisting them in controlling this infection, as well as other poultry diseases, and also that if possible opportunities should be opened for veterinarians who previously have not been more than passively interested in poultry practice to become better developed in this line of endeavor. Dr. G. R. Dunlap exhibited a ques-

tionnaire which had been used to advantage in Illinois in accumulating information in regard to the local veterinary practitioners' reaction toward poultry practice and their willingness to participate in that line of endeavor.

Good suggestions were made by several of those present regarding how the interest may be sustained over a period of two or three years in horse parasite control programs.

Dr. Gilbert S. Weaver introduced a questionnaire which he proposed to use in his state to see if more accurate statistics regarding animal morbidity and mortality could not be obtained. This elicited considerable discussion regarding the percentage of replies obtained from questionnaires and the best possible ways of making use of such replies.

Several speakers indicated that if a general plan for a veterinary extension program could be devised, it would be desirable as a guide for future endeavors, although it was acknowledged that the conditions in each state would have to be taken into consideration in applying such a plan.

Members' suggestions regarding next year's meeting indicated the desirability of having speakers assigned to certain topics and then limiting the time for the discussion of each topic. The possibility of covering more topics of interest to the group might be accomplished in this way.

Eighteen More 1937 Graduates

At the close of the summer session, at Kansas State College, July 30, 1937, the degree of Doctor of Veterinary Medicine was conferred on the following:

Ben Salvatore Burdo — Maurice Crouch
Robert George Cotten — Edmund Burke McCormick
Carl Fred Steinhauser

The following students were graduated from the School of Veterinary Medicine, Alabama Polytechnic Institute, at the close of the summer session, August 20, 1937:

William Elmer Donavan — LaRua Garrett
Richard Sylvester Duffell — Elton McLaurin Spence
Fred Thompson

At the commencement exercises held by the Colorado State College, on August 20, 1937, the degree of Doctor of Veterinary Medicine was conferred upon:

John Thimmig

The following veterinary students were graduated at the summer school commencement of the Agricultural and Mechanical College of Texas, August 28, 1937:

N. A. Cox
Henry Donelson
Robert Elmore Erisman
J. Y. Henderson
Kermit Helmer Hoyme
John B. Reneau, Jr.

At the close of the summer session, Michigan State College conferred the D. V. M. degree on

L. E. Jameson

These 18 additional graduates bring the 1937 total up to 304.

World's Horse-Pulling Record

A new world's record for a horse-pulling contest was established at the Ohio State Fair, on September 1, 1937, when Jerry and Barney, a team of Belgians, made a tractive pull of 3,925 pounds the full distance of 27½ feet. This is equivalent to starting, for 15 to 20 consecutive times, a gross load of 50,960

JERRY AND BARNEY IN ACTION

pounds on granite block pavement, on a wagon. The previous record was 3,900 pounds. The team is owned by Dr. W. M. Crall and Thomas Gamboe, of Pioneer, Ohio. When the team went into action at the Ohio State Fair, it was the first time that the two horses had ever pulled together. They were entered in the horse-pulling contests at the Indiana State Fair, and at the Chicago Charter Jubilee.

Regular Army

Lt. Colonel Harry J. Juzek is detailed for duty as attending veterinarian, Portland, Oregon, for the purpose of inspecting perishable items purchased on contract by the Quartermaster of the San Francisco General Depot and Seattle Quartermaster Depot, in addition to his other duties at Vancouver Barracks, Wash., and will report by letter to the Commanding General, San Francisco Port of Embarkation, Fort Mason, Calif., for instructions.

Major Nathan M. Neate is directed to proceed to Carlisle Barracks, Pa., and report to the Commandant, Medical Field Service School, on October 14, 1937, for temporary duty until about December 19, 1937, for the purpose of pursuing the advanced course of instruction; upon completion of this duty to return to his proper station, Langley Field, Va.

Major John K. McConeghy having been examined for promotion by a board of officers and found physically disqualified for the duties of a lieutenant colonel of the Veterinary Corps, by reason of disability incident to the service, his retirement by the President from active service as a lieutenant colonel under the provisions of the act of Congress approved 23 April, 1908, and the act of Congress approved 23 April, 1930, is announced, to become effective 30 September, 1937, with rank from 3 September, 1937, the date upon which he would have been promoted to that grade by reason of length of commissioned service if found qualified.

The appointment of Captain Earl Goss Kingdon, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from October 1, 1937, and his assignment to duty at Fort Bliss, Texas, is announced.

The promotion of the following-named officers to rank from date opposite their names is announced.

To Colonel

Lt. Colonel George H. Koon, September 27
Lt. Colonel Daniel B. Leininger, September 29

To Lieutenant Colonel

Major Francois H. K. Reynolds, Sept. 3
Major Sawyer A. Grover, Sept. 10
Major Charles S. Williams, Sept. 10
Major Fred W. Shinn, Sept. 10
Major Philip H. Riedel, Sept. 10
Major Irby R. Pollard, Sept. 10
Major Frank C. Hershberger, Sept. 10
Major Clifford E. Pickering, Sept. 13

Major Frank B. Steinkolk, Sept. 13
Major Raymond Randall, Sept. 14
Major Seth C. Dildine, Sept. 16
Major Joseph H. Dornblaser, Sept. 16
Major George L. Caldwell, Sept. 18
Major Jacob L. Hartman, Sept. 18
Major John H. Kintner, Sept. 18
Major Samuel G. Kielsmeier, Sept. 21
Major Peter T. Carpenter, Sept. 22
Major Oness H. Dixon, Jr., Sept. 26
Major John W. Miner, Sept. 29

Veterinary Reserve Corps

PROMOTIONS

	To	
Bechtol, Lauren Leonhardt	Capt.	123 E. Butler St., Bryan, Ohio.
Galloway, Walter Gilmore	Capt.	R. R. No. 2, West Lafayette, Ind.
Kingdon, Earl Goss	Capt.	1327 S. 14th East, Salt Lake City, Utah.
Morgan, Donald Richard	Capt.	402 E. 29th St., Vancouver, Wash.

NEW ACCEPTANCES

Chastain, Frank Loyd	1st Lt.	Colbert, Ga.
Gochenour, Wm. Sylva, Jr.	1st Lt.	705 Varnum St., N. W., Washington, D. C.
Moss, Lyle Alonzo	1st Lt.	Room 218 Federal Bldg., Sacramento, Calif.
Steele, Walter Howard	1st Lt.	2115 N. E. 38th Ave., Portland, Ore.
Turner, Clifford Wesley	1st Lt.	Broomfield, Colo.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Hervey, William Hugh	1st Lt.	Fort Leavenworth, Kan.
Paul, Emmet W.	1st Lt.	Fort Douglas, Utah.
Watson, Edward J.	1st Lt.	Los Angeles, Calif.
Lancaster, Harry Ruyle	1st Lt.	Fort Benning, Ga.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Phifer, William Gough	1st Lt.	Camp Beauregard, La.
Warnock, Robert S.	1st Lt.	Fort Brady, Mich.
Adams, John D.	Capt.	Boise, Idaho.
Castleberry, Guy	Capt.	Fort Douglas, Utah.
Mendenhall, William I.	1st Lt.	Pocatello, Idaho.
Chastain, W. R.	Capt.	Fort Benning Ga.
Whitfield, John S.	1st Lt.	Fort Oglethorpe, Ga.
Cairy, Clyde Frank	1st Lt.	Fort Lincoln, N. D.
Rippetoe, Culver Willis	1st Lt.	Fort Leavenworth, Kan.
Smit, Charles Rudolph	1st Lt.	Fort Lincoln, N. D.
Wiswell, Wilbur Harold	1st Lt.	Fort Lincoln, N. D.

The Royal Winter Fair Horse Show will be held in Toronto, Canada, November 16-24.

NECROLOGY

GARRY N. WICKWIRE

Dr. Garry N. Wickwire died at his home in Indianapolis, Ind., on August 25, 1937. He had been in poor health since his retirement from active service in the U. S. Bureau of Animal Industry about three years ago.

Born in Angola, Ind., December 25, 1861, Dr. Wickwire received his early education in the public schools of that city. Later he attended the Tri-State College, in Angola, and was graduated with the first class in 1886 with the degree of B. S. He later studied dentistry and was graduated from the Indiana Dental College in 1897. Some years later, he decided to study veterinary medicine and entered the Indiana Veterinary College. Following his graduation in 1908, he received an appointment in the U. S. Bureau of Animal Industry and was assigned to meat inspection at Newark, N. J. Two years later, he was assigned to tick eradication work in Mississippi. In 1914, he was assigned to hog cholera control work and was stationed at various times in Iowa, Kansas, Indiana and Kentucky. In 1920, he was transferred to Indiana, where he remained until his retirement in 1934.

Dr. Wickwire joined the A. V. M. A. in 1920. He was a member of the National Association of Bureau of Animal Industry Veterinarians and the Masonic Lodge of Angola. He held the distinction of having been a member of the latter organization for 51 years. He is survived by his widow (née Janette Byers), one daughter and two sisters.

EDWARD S. GREENWOOD

Dr. Edward S. Greenwood, who formerly was located at Laurens, Iowa, died in the Cherokee, Iowa, Hospital, July 20, 1937, in his 59th year. Dr. Greenwood was a graduate of the Ontario Veterinary College, class of 1905, and of the McKillip Veterinary College, class of 1912. He joined the A. V. M. A. in 1916.

PHILLIP M. JAMES

Dr. Phillip M. James, of Amarillo, Texas, died at home on June 5, 1937, following an acute heart attack. He had been in poor health for some time and recently had undergone a major operation. He was a graduate of the Kansas City Veterinary College, class of 1906, and was in the employ of the U. S. Bureau of Animal Industry. He had been stationed in Texas since 1917, on tick eradication and quarantine work. He was a member of the National Association of B. A. I. Veterinarians.

WILLIAM J. SCANLON

Dr. William J. Scanlon of Kansas City, Kan., died of pneumonia in Wesley Hospital, Kansas City, Mo., on July 26, 1937. He was 50 years old.

Born in Chapman, Kan., Dr. Scanlon received his early education in the local schools and then entered Kansas State College for the study of veterinary medicine. He was graduated in 1915 and the same year entered the service of the U. S. Bureau of Animal Industry. His first assignment was on meat inspection at Sioux Falls, S. Dak.

Dr. Scanlon joined the A. V. M. A. in 1918. He was a member of the National Association of B. A. I. Veterinarians. He is survived by one son, one daughter, three brothers and a sister.

O. A. DIVENS

Dr. O. A. Divens, of Good Hope, Ohio, died at his home, August 23, 1937, at the age of 65 years. He had been ill for quite some time. He was a graduate of the Terre Haute Veterinary College, class of 1913, and practiced in Fayette County for many years. He is survived by his widow and one brother.

DAVID W. CURTIS

Dr. David W. Curtis, of Cadillac, Mich., died at his home, September 5, 1937, following a heart attack. He had been ill for about a month.

Born in Leamington, Ontario, January 20, 1863, Dr. Curtis was graduated from the Ontario Veterinary College in 1892. He practiced for brief periods at Big Rapids and Reed City, Mich., before locating in Cadillac in 1893.

Dr. Curtis was a member of the Michigan State Veterinary Medical Association. He is survived by one daughter. Burial was at Listowell, Ont.

WILLIAM WALLACE EAGLE

Dr. William W. Eagle died at his home in Kansas City, Kan., September 10, 1937, after an illness which had confined him to his bed for over three months. He had been in the service of the U. S. Bureau of Animal Industry for several years, having been reinstated during the recent drouth and assigned to appraisal work.

DR. W. W. EAGLE

Born in Kansas City, Kan., February 9, 1884, Dr. Eagle attended local schools and was graduated from the Kansas City Veterinary College in 1906. Shortly thereafter, with two brothers, Dr. R. M. Eagle and Dr. John Eagle (K. C. V. C. '04), both now deceased, and a brother-in-law, Dr. R. B. Crimes (K. C. V. C. '05), Dr. Eagle organized the Eagle Serum Company, which remained in existence for about 20 years. The property was sold last year to a packing company.

Dr. Eagle joined the A. V. M. A. in 1909. He was a member of Albert Pike Masonic Lodge; Oriental Commandery, Knights Templar, and Ararat Temple of the Shrine, of Kansas City, Mo., and of Caswell Consistory, Scottish Rite, of Kansas City, Kan. He is survived by his widow (née Mildred Morrison) and two

brothers, Dr. R. F. Eagle (K. C. V. C. '01), of Chicago, a vice-president of Wilson and Company, and Dr. Thomas J. Eagle (K. C. V. C. '03), of Albany, Mo.

GEORGE LELAND ESTEY

Dr. George L. Estey, of Eden Valley, Minn., succumbed to a heart attack, September 26, 1937.

Born at Royalton, Minn., September 16, 1892, Dr. Estey attended local grade and high schools, before entering the Kansas City Veterinary College. Following his graduation in 1916, he located in Eden Valley and practiced there until his death, except for a period of about six months, during the World War, when he was stationed at the Veterinary Training School at Camp Lee, Va., as a second lieutenant in the Veterinary Corps of the Army. Just prior to his discharge, January 20, 1919, he was promoted to the rank of captain.

Dr. Estey joined the A. V. M. A. in 1924. He was a member of the Minnesota State Veterinary Medical Society, the American Legion Post of Eden Valley No. 381, the Masonic order and the Methodist Episcopal Church. He is survived by his widow (née Helen Litcher Hick) and one sister.

DAVID F. SHAKE

Dr. David F. Shake, of Carlisle, Ind., died in the Sherman Hospital, Sullivan, Ind., October 11, 1937, at the age of 74.

Born in Warren County, Iowa, Dr. Shake moved to Indiana at an early age. He was a graduate of the Indiana Veterinary College, class of 1902, and had practiced at Carlisle for more than 30 years. He is survived by his widow, three sons and four brothers.

SAMUEL CROUCH

Dr. Samuel Crouch, of Altadena, Calif., died in the Los Angeles General Hospital, October 18, 1937, of rabies. On July 19, Dr. Crouch was bitten by a dog which died the following day. A positive diagnosis of rabies was made by the Pasadena Health Department. On July 20, Dr. Crouch had his wounds cauterized and he immediately started taking a series of injections of rabies vaccine. On October 11, he showed signs of illness which were thought at first to be due to neuritis. October 12 and 13, he

experienced difficulty in swallowing, in addition to extreme discomfort in both arms. On October 14, a physician ordered him to the hospital, as he was of the opinion that the condition was rabies. Four days later, Dr. Crouch died and the laboratory later confirmed the diagnosis.

Born June 21, 1879, in Henderson County, Ill., Dr. Crouch studied veterinary medicine at the McKillip Veterinary College. He was graduated in 1920 and first practiced in Klamath Falls, Ore. He remained there for several years and then moved to southern California. He practiced in Los Angeles, Glendale and Pasadena before locating in Altadena.

Dr. Crouch joined the A. V. M. A. in 1929. He is survived by his widow, one stepson, one brother and four sisters.

W. L. C.

SIMON FRASER TOLMIE

Dr. S. F. Tolmie, of Victoria, British Columbia, a former premier of the province, Minister of Agriculture in two federal cabinets, and five times member for Victoria in the House of Commons, died October 13, 1937, after a long illness.

Born at Cloverdale, near Victoria, B. C., January 25, 1867, Dr. Tolmie attended local schools, was graduated from the Victoria High School and entered the Ontario Veterinary College. Following his graduation in 1891, Dr. Tolmie returned to British Columbia and started to practice in Victoria. A short time later, he was appointed Inspector of Animals at Victoria, for which work he received the modest salary of $7.00 per week. He was soon offered the position of Inspector of Animals for the British Columbia government, and later the Dominion Department of Agriculture sought his services. The provincial government was reluctant to lose him and an arrangement was effected by which he worked for both. He enjoyed the unique distinction of being retained by the Conservative government at Ottawa and by the Liberal government at Victoria. A short time later, Dr. Tolmie resigned his provincial post and became Chief Inspector of Animals for the federal Department of Agriculture.

When Rt. Hon. Arthur Meighen succeeded Sir Robert Borden on the latter's resignation as war-time Prime Minister, Mr. Meighen recognized in Dr. Tolmie the agricultural ability that would fit him for the duties of Minister of Agriculture. During his incumbency in this office he won the respect and admiration of all parties. Again, in 1926, when Mr. Meighen came into

power for the second time, he appointed Dr. Tolmie as Minister of Agriculture.

In 1926, the Conservative Party of British Columbia drafted Dr. Tolmie to become the party leader in that province. He was elected Premier of British Columbia in 1928, but was defeated in 1933, when he went to the polls with a Unionist government. This was the sole reverse of his political career, during which he was elected five times as member for Victoria in the House of Commons at Ottawa.

Dr. Tolmie joined the A. V. M. A. in 1901, and at the time of his death was one of the oldest members on the roll. He was a charter member of the British Columbia Veterinary Association and had served as both president and secretary of the organization. According to Dr. W. Graham Gillam, secretary and registrar of the British Columbia Veterinary Association, Dr. Tolmie retained his interest and membership in the organization throughout his long and arduous political career.

Dr. Tolmie served as president of the British Columbia Agricultural Association and as an officer and counselor in a score of other live stock and farm organizations. He had marked ability as a breeder and judge of live stock. He is survived by two sons, two daughters, one brother and one sister.

DAVID HENDRICKS BERGEY

Dr. David H. Bergey, formerly professor of bacteriology and hygiene at the University of Pennsylvania, died September 5, 1937, at the age of 76 years. Dr. Bergey was well known to many of the older veterinary graduates of the University of Pennsylvania, as he was demonstrator (1902-03) and assistant professor (1903-10) of bacteriology in the School of Veterinary Medicine. His service to the University of Pennsylvania started in 1893, when he was appointed as a fellow in hygiene. He terminated his service in the post of Director of the Laboratory of Hygiene, in 1932, to become director of research in biology of the National Drug Company, of Philadelphia. Undoubtedly Dr. Bergey's most lasting contribution to science is his "Manual of Determinative Bacteriology," the fifth edition of which is now in press. His "Principles of Hygiene" passed through seven editions.

Our sympathy goes to Dr. Lawrence A. Mosher, of Atlanta, Ga., in the death of his wife, September 25, 1937, following a heart attack.

PERSONALS

MARRIAGES

DR. VIRGIL A. YOUNT (Colo. '35), of Kansas City, Kan., to Miss Mitzy Hart, of Durant, Okla., at Cherokee, Okla., May 2, 1937.

DR. EDWARD A. MURPHY (K. S. C. '36), of Omaha, Neb., to Miss Louise Ratliff, at Manhattan, Kan., June 14, 1937.

DR. MARVIN J. TWIEHAUS (K. S. C. '36), of Manhattan, Kan., to Miss Dorothy Washington, at Manhattan, September 4, 1937.

DR. PHILIP HENRY GERLACH (U. P. '37), of Milford, Conn., to Miss Doris Antoinette Oswald, August 26, 1937, at New York, N. Y.

DR. FLOYD E. MONROE (K. S. C. '37), of Phoebus, Va., to Miss Helen McCord, of Topeka, Kan., at Topeka, September 3, 1937.

BIRTH

To DR. and MRS. BERNARD H. GIBSON, of Newark, Ohio, a girl, Kathryn Jane, October 20, 1937.

PERSONALS

DR. JAMES L. BOYDSTON (Mich. '37) recently located at Athens, Mich., for general practice.

DR. E. W. WILSON (K. S. C. '31), who has been practicing at Glover, Vt., has removed to Newport, Vt.

DR. REUBEN HILTY (Ohio '07), of Toledo, Ohio, is president of the Board of Commissioners of Lucas County.

DR. JAMES R. KETCHERSID (K. S. C. '37) is now associated with Dr. Charles W. Bower (K. S. C. '18), of Topeka, Kan.

DR. JOHN J. ARNOLD (Mich. '37) has been appointed as an instructor in surgery and medicine at Michigan State College.

DR. R. C. RIEGEL (Gr. Rap. '15), of Harrisburg, Ill., was hurt in an automobile accident, about the middle of September.

DR. JOHN E. KELLBERG (U. P. '35) recently opened a new hospital on U. S. Route 6, about two miles east of Princeton, Ill.

DR. R. V. SMITH (Ont. '88), of Frederick, Md., has been on the sick list for several weeks and is now confined to his home.

DR. E. C. GOODRICH (Gr. Rap. '15), who has been located in Louisville, Ky., for some time has returned to Kalamazoo, Mich.

DR. EWING M. JOHNSON (Iowa '08), of Chicago, Ill., was retired from active service in the U. S. Bureau of Animal Industry recently.

DR. LOUISE SKLAR (K. S. C. '34), is taking graduate work in the Department of Veterinary Science at the University of Maryland.

DR. CHARLES H. ERGANBRIGHT (Ont. '92), who has been located at Flat Rock, Ind., for the past 20 years, has moved to Indianapolis.

Dr. EDMUND E. VISGER (Mich. '37) has received an appointment as instructor in anatomy, Division of Veterinary Science, Michigan State College.

DR. CHARLES K. FRANCIS (Ont. '37), has accepted a position with the Maryland Live Stock Sanitary Board and is now stationed at College Park, Md.

DR. AARON NEWBURG (K. C. V. C. '09), who was on the Kansas City, Kan. force of the U. S. Bureau of Animal Industry was retired recently from active service.

DR. F. E. CASWELL (Ont. '13), of North Adams, Mich., has opened a drug store in his village. It is being operated as a separate business, apart from his practice.

DR. LLOYD D. JONES (Iowa '31), who has been on active duty in the Veterinary Reserve Corps, at Camp Bewabis, Crystal Falls, Mich., is now located in Winnetka, Ill.

DR. ROLLAND SLAUGHENHAUPT (Mich. '36) has resigned from the service of the U. S. Bureau of Animal Industry and has entered general practice at Albion, Mich.

DR. ROBERT H. WILSON (K. S. C. '09), of Rochester, Mich., addressed the local Kiwanis Club on September 14. His subject was "The Progress of Veterinary Medicine."

DR. J. L. AXBY (Chi. '03), of Indianapolis, Ind., addressed the Kiwanis Club of Greensburg, Ind., on October 12. He gave a vivid interpretation of his philosophy of life.

DR. WENDELL E. DAVIS (Mich. '34) has left the employ of the Detroit City Board of Health and has taken over the practice of the late Dr. James M. Miller, at Benton Harbor, Mich.

DR. E. D. RHOADES (K. C. V. C. '15) has resumed his practice at Lincoln, Ill., after spending five months in the service of the U. S. Bureau of Animal Industry in Wisconsin.

DR. JOHN F. RYFF (Mich. '35) has given up his practice at Caledonia, Mich., to become an instructor in Bacteriology at Michigan State College.

DR. JAMES W. BENNER (K. S. C. '11), formerly at the New York State Veterinary College, at Cornell University, Ithaca, N. Y., has requested a change of address to State College, N. Mex.

DR. J. W. SCALES (Mich. '34) has been appointed assistant instructor in biology and animal pathology at the Virginia Polytechnic Institute, Blacksburg. He assumed his duties September 18.

DR. JAMES R. CORLISS (K. C. V. C. '15), of Newport, Vt., has given up his practice at that point and has returned to Maine, where he is again employed by the U. S. Bureau of Animal Industry.

DR. C. L. CAMPBELL (O. S. U. '26) resigned his position with the Haver Glover Laboratories, of Kansas City, Mo., September 1, and has accepted a similar position with the Lederle Laboratories.

DR. WM. T. S. THORP (Mich. '35), formerly pathologist to the Michigan Department of Agriculture, is now an instructor in animal pathology in the Division of Veterinary Science, Michigan State College.

DR. C. H. COLTON (Colo. '21), has resigned from the service of the California Division of Animal Industry and is conducting the Estado Pet Clinic, at Santa Barbara, Calif., which formerly was known as the Nelson Pet Clinic.

DR. N. S. MAYO (Chi. '89), of Highland Park, Ill., accompanied by Mrs. Mayo, started on a trip, October 14, which will take them to Cuba, Panama and Central America. They expect to return during the first week of November.

DR. T. E. MONTGOMERY (Gr. Rap. '12) has been dismissed as superintendent of live stock at the Jackson, Mich., prison, following the abolishment of the office. In the future the services of local practitioners will be used at the prison farm. Dr. Montgomery has opened a hospital for small animals in Jackson.